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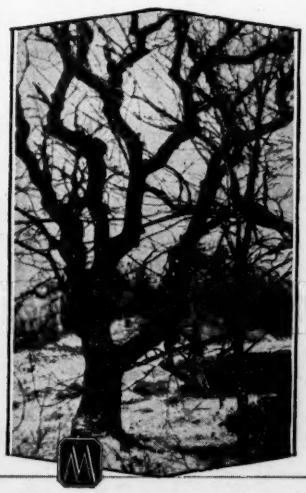

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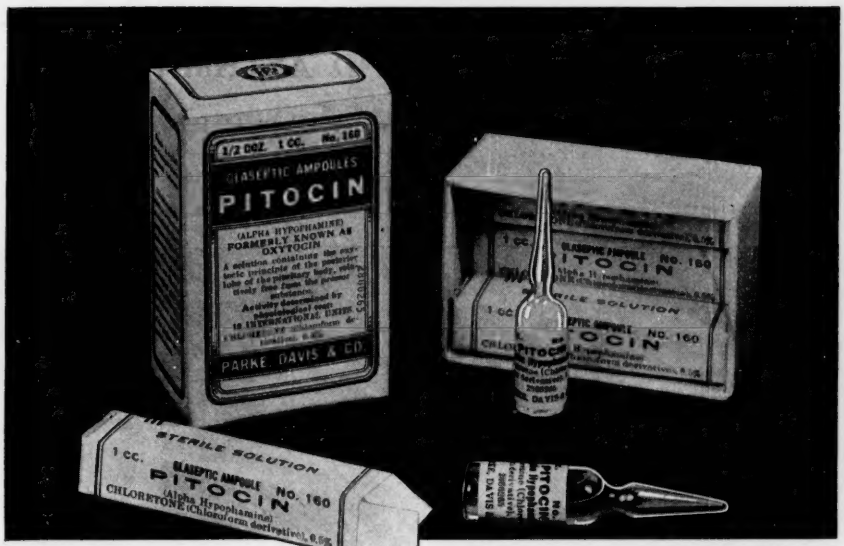

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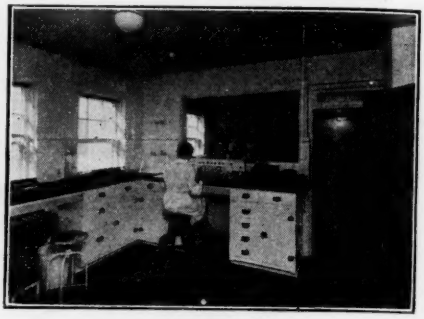

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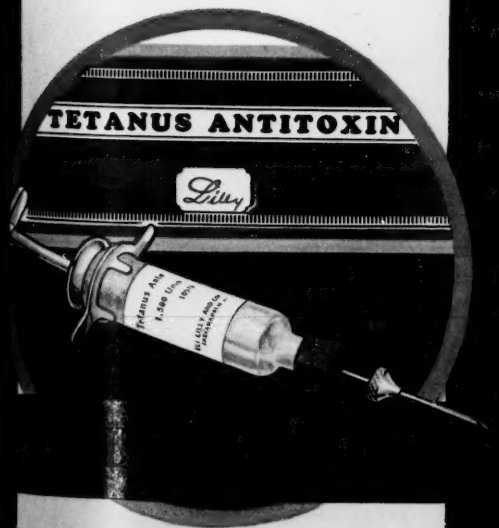

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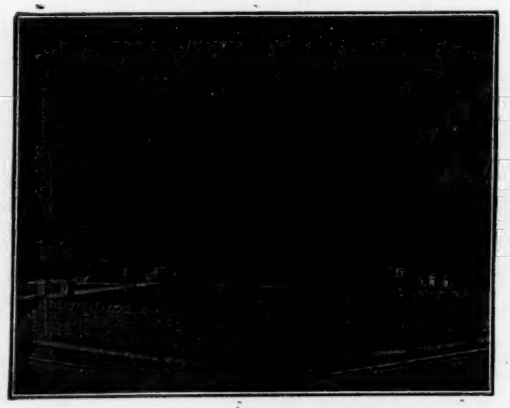

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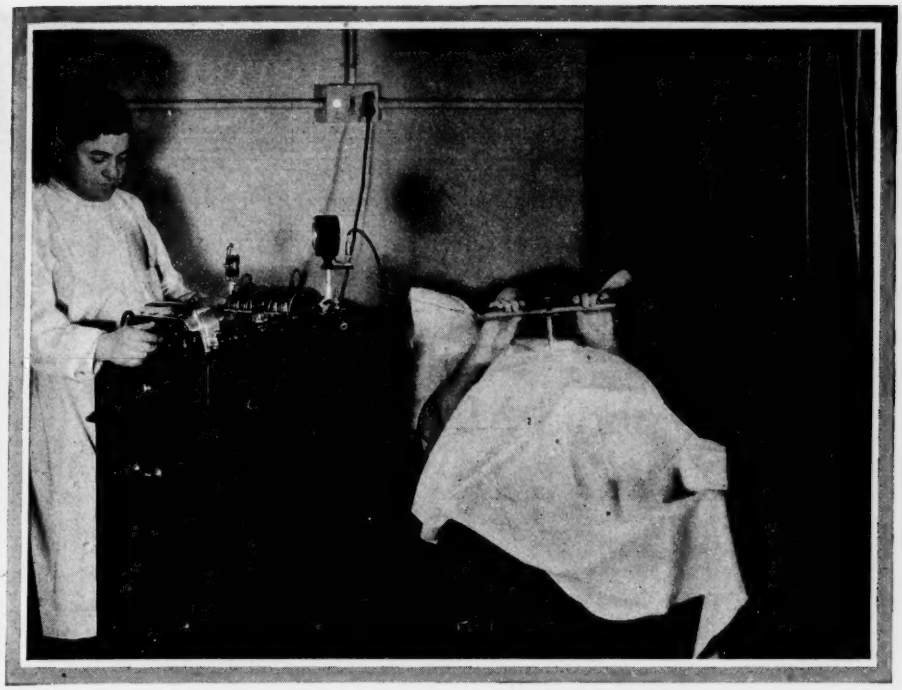

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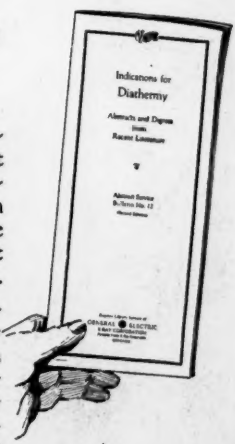

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Congenital Heart Disease—Case of Tetralogy of Fallot

Don Carlos Peete, M.D., Kansas City.
Department of Medicine, University of Kansas.

A very interesting patient recently came to the Eye Clinic, complaining of headaches and increased watering of the eyes. The pronounced clubbing of his fingers was noted by Dr. John Billingsley, who referred the patient to the Medical Clinic for examination. The patient is an undersized colored man, 22 years old, 4 feet 11 inches in height, weighing 69.5 pounds.

Fig. I

C. C. Headaches, dizziness, shortness of breath, cough.

P. I. After working as a clerk in a grocery store until September, 1929, he was forced by increasing weakness, a cough and dyspnea to go to bed for a month. He was told that his illness was due to his heart. Since recovery from this illness he has not regained his former strength, is unable to walk more than two blocks without much difficulty and respiratory embarrassment; headaches and dizziness, cough and dyspnea are very marked on exertion.

P. H. The patient had much sickness during early childhood, the usual childhood diseases seemed to be much more severe for him than other children in the family. He says he was carried about on a pillow when a baby; unable to run and play games with other children because of dyspnea and cough; had measles in 1925, very severe; had penile sore in 1921 which was treated by circumcision. Finished high school at 17 years, was unable to take cadet training due to dyspnea and weakness on exertion.

F. H. Father died in 1922 at 42 years of age. Mother died in 1925 at 32 years, cause unknown. Three brothers and one sister living and well. The patient was a third child, one brother, several years younger and much larger, is able to carry him on his back.

Physical Examination: Patient, a small colored man, appears to be about 14 years old, but actually is 22 years of age; has a marked clubbing of fingers and toes; a deep purple hue of mucous membranes is noted. The pupils react to light and accomodation. The tongue, lips and gums have the dark, purple appearance such as left after eating blackberries. Pulsating vessels are seen in the neck, probably carotids.

There is a mild enlargement of left chest but practically no scolioses. A definite cardiac impulse is felt at the precordial area but no definite thrill is made out. The P.M.I. is diffused and

can be seen in the seventh interspace and as far as 12 cms. to the left of the mid-sternal line; R.C.D. to the left 10 cms. and 4 cms. to right. There is a loud

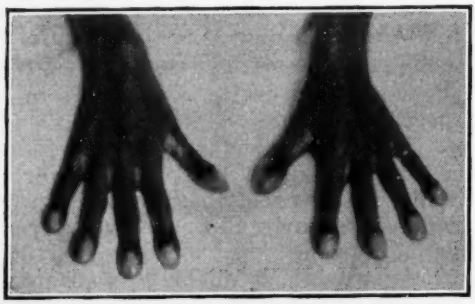

Fig. II

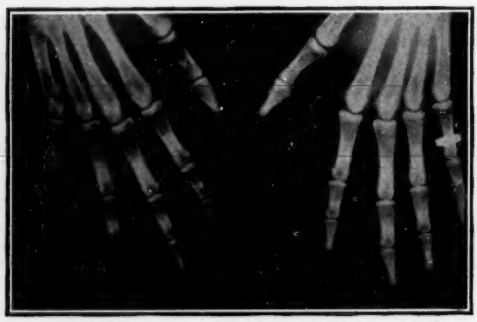

Fig. III

diastolic murmur heard over the entire precordium which is loudest at the 4th interspace to the left of the sternum, the murmur being transmitted to the vessels of the neck, but not transmited to the axilla; may be heard faintly to the left of the 2nd and 3rd vertebrae posteriorly. There is a snappy first sound at the apex which sounds greatly like a presystolic or an Austin Flint murmur. A 2 is great than P 2. A systolic murmur is heard over the base in the 3rd interspace to the left of the sternum.

Pulse: rate 20 to ¼ minute, regular, poor volume and tension. Blood pressure not obtainable with the Baumanometer. Recording Sphygmomanometer shows it, with a very feeble excursion, to be systolic 70, diostolic 35. Pachon's oscillometer, systolic 90, diastolic 50. Moist rales are heard at each base.

Liver is palpable 2½ cms. below costal margin. Abdomen otherwise negative. K.K. active; feet show clubbing of toes, no edema.

Laboratory Reports: Wassermann and Kahn negative. Blood examination: Hgb. 123%; red cells 9,104,000; leucocytes 4,400; color index 0.6; fresh blood black and viscous; P.M.N. 59%; P.M.E. 1%; S.M. 39%; transitionals 1%. Urine examination, straw, acid, 4 plus albumin, specific gravity 1.022, negative sugar, many granular casts.

x-Ray report by Dr. John Rodick is as follows: "Examination of the chest shows the diaphragm smoothly contoured, although the left is somewhat elevated. The hilar shadows and radial markings are moderately increased but there is no evidence of infection in the lungs.

"The heart shadow measures 3¾ cm. to the right of the midline and 10 cm. to the left. This shows a total width of 13¾ cm. while half the inside diameter of the chest is but 11½ cm., thus demonstrating enlargement of the heart with the greatest change in the left ventricle. Fluoroscopic examination confirms these findings and, although there is some widening of the aorta, the retro-cardiac space is not decreased by any appreciable enlargement of the left auricle and there is no abnormal prominence of the pulmonary conus."

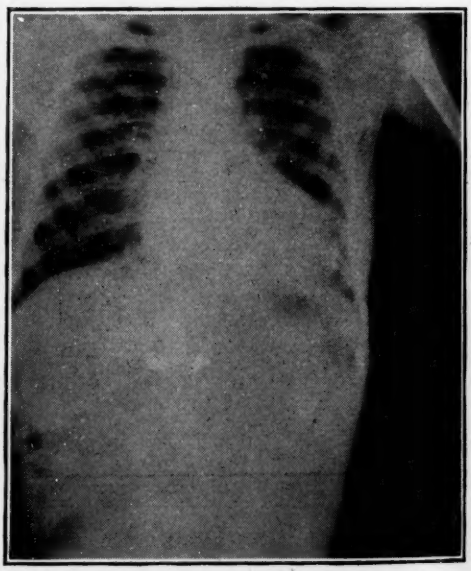

Fig. IV

"The left ventricular enlargement appears to be the predominant lesion and produces the cardiac contour commonly seen in aortic stenosis and regurgitation of moderate degree."

Electrocardiogram: the P wave is increased in amplitude, and there is also evidence of right ventricular preponderance.

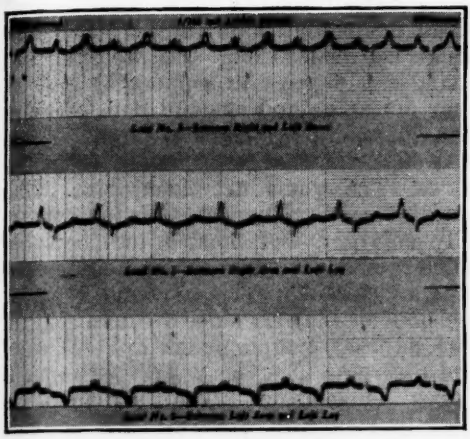

Fig. V

March 5, 193. Patient has been bleeding from gums.

March 8, 1930. Dizzy headaches.

March 14, 1930. Had an epileptiform seizure 5 days ago, after having walked a mile.

SUMMARY

1. Headaches—dizziness—epileptiform seizures.
2. Marked retardation in growth.
3. Clubbed fingers and toes.
4. Deep cyanosis.
5. Enlarged heart, *x*-ray evidence of left sided hypertrophy.
6. Electrocradiographic evidence of right ventricular preponderance.
7. Loud diastolic murmur faintly transmitted to vessels of neck and to back, but not to axilla.
8. Rales at base of lungs.
9. Liver 2½ cms. below costal margin.
10. R.B.C. 9,104,000, H.P. 123%.
11. Albumin 4 plus, granular casts.

DIAGNOSIS (TENTATIVE)

1. Congenital heart disease.
2. Pulmonic stenosis.
3. Intraventricular defect.
4. Dextra position of the aorta.
5. Right ventricular hypertrophy.
6. Left ventricular hypertrophy.
7. Aortic insufficiency.

As we are yet unable to report any necropsy findings in this case, our diagnosis, in some respects, must be more or less speculative, and for that reason it has brought out some very interesting discussion. About 50 per cent of all congenital heart cases are found to have either a pulmonary stenosis or an atresia. Most of these cases show an intraventricular defect, and many show the aorta arising above the defect from both the right and left ventricles. Fallot was the first to call attention to the clinical and pathological picture of the most common type of congenital heart disease seen. There are four outstanding anatomical findings which are associated with such cases called "the tetralogy of Fallot," *i.e.*; 1. Pulmonary stenosis. 2. Intraventricular septal defect. 3. Right ventricular hypertrophy. 4. Dextra position of the aorta.

Most all who have seen this patient are agreed that there is a pulmonary stenosis, an intraventricular septal defect, and we have electrocardiographic evidence of a right sided hypertrophy. Now, as to the dextra position of the aorta, we have no means of proof except that, as Fallot and others pointed out, it is one of the usual anatomical findings.

Is there an aortic insufficiency in this case? There has been much discussion on the question, some of which follows. There is a loud diastolic murmur heard best at the 4th interspace to the left of the sternum, a questionable Austin Flint murmur, throbbing carotids, and *x*-ray evidence of left sided hypertrophy. The blood pressure in this case would tend to disprove aortic disease, but we can hardly compare normal blood pressure phenomena with that of a congenital heart, especially in a heart that has such poor tone. The cause of this aortic insufficiency, if it does exist, is also open to discussion. Abbott has shown the frequent association of subacute bacterial endocarditis with congenital heart disease. There is also a possibility of luetic aortitis with regurgitation. This patient gives a history of a primary lesion. The

blood Wassermann and Kahn are negative, but, we frequently find that phenomena occurring in cases that show luetic aortitis at necropsy.

Dr. Ralph Major and Dr. Thomas G. Orr reported a patient at the staff meeting in April, 1929, having a patent foramen ovale with an aneurysm of both right and left branches of the pulmonary artery which was anatomically luetic. The blood Wassermann and physical examination for lues were negative.

Finally: This is a report of a patient with congenital heart disease, in which we make the diagnosis of the tetralogy of Fallot with aortic regurgitation. Necropsy findings will be of great interest and may show us to be wrong in our conception of existing conditions.

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Generalized Tuberculous Lymphadenitis

SAMUEL D. KATZ, M.D.
Department of Pathology

Tuberculosis of the lymph nodes many times simulate, clinically, other diseases of the blood forming organs. The following is a case which clinically was diagnosed Hodgkin's Disease or (?) Lymphosarcoma, but autopsy findings were those of hyperplastic tuberculous lymphadenitis.

G. K. 13 months old, white male, who was brought to Bell Memorial Hospital on October 1, 1929. History of feeding difficulty at 3 months of age was obtained. He was taken off of breast milk and evaporated milk was substituted. He continued to be no better and it was noticed that the glands in his neck began to enlarge. Family history of scrofula (tuberculosis) in father.

Physical Examination: The child appeared emaciated and anemic. He showed papular eruptions on the soles of the feet and marked enlargement of the anterior and posterior cervical lymph glands, axillary and inguinal glands. Child on admission did not appear very ill though at times later became very restless and cried very much. W.B.C. count 17,000.

Progress: October 12, 1929, some glands of the neck were removed for examination. Following the operation, the child continued to become more restless and died October 13, 1929, at 11:10 a. m.

AUTOPSY

General Inspection: The body is that of an emaciated male infant measuring about 24 inches long and weighing 13 pounds. He appears emaciated and of poor muscular and skeletal development. The anterior fontanelles are open. There is a slight discharge of mucus from the nares. The gums are edentulous. The anterior cervical lymph nodes are markedly enlarged and matted together, being larger superiorly and smaller toward the clavicles. There is a recent surgical incision on the left side, 3 cm in length. The axillary lymph nodes are enlarged and discrete. The chest wall is thin and shows emaciation. The abdomen is slightly rounded. Several firm nodules can be felt throughout the abdomen. No organs are palpable. The inguinal lymph nodes are enlarged.

Peritoneal Cavity: Peritoneal surface shows small granulations, which are opaque in color. The small and large intestines are matted together by the enlarged, nodular mesenteric lymph nodes and adhesions.

Thoracic Cavity: Adhesions are found between the middle lobe of the right lung and the chest wall. Anterior mediastinal lymph nodes, and those along the internal mammary vessels are enlarged.

Organs: The lungs weigh 110 grams. Adhesions are present on the surface. Small grayish white tubercles are also seen. Cut surface shows some congestion as well as small tubercles. Bronchi contain thick mucus. The tracheobronchial lymph nodes are enlarged, and on cut section show caseation. Liver and spleen show small tubercles over the surface, and on cut section.

Thymus, heart, pancreas, gall bladder, adrenals and kidneys show nothing abnormal.

The left scrotum is enlarged, showing an edematous condition of the tunica vaginalis and cystic hydrocele.

Small intestines are filled with greenish fecal material. The serosal surface is studded with small grayish-white nodules measuring from .5 to 1 mm. in diameter. In certain segments clusters of these tubercles are seen on the peritoneal surface. Section through these patches show typical ragged ulcers, tending to circumscribe the gut. The cecum is surrounded by enlarged lymph nodes. Transverse and descending colon show the same serosal appearance. The mesenteric lymph nodes are markedly enlarged to 3 to 4 cm. in diameter. They are matted together with other glands, though at places are discrete. The mesentery is thickened and is covered with patches of tubercles. The glands cut with a gritty sensation and on cut section show a dense white fibrous appearance, though occasionally a gland presents a yellow caseous-like material. Cut section of the other enlarged lymph glands presents a similar appearance.

HISTOLOGICAL PATHOLOGY

Lungs, liver and spleen present a typical picture of miliary and conglomerate tuberculosis, with a small amount of caseation. Thymus, heart, pancreas, adrenals and kidneys show nothing of an abnormal nature.

Sections of small intestines show a large number of well developed but small miliary tubercles on the peritoneal surface. Some tubercles are seen forming a portion of the wall and base of an ulcer. A similar change in the appendix is seen.

Sections of the lymph glands show the lymphoid tissue to be largely replaced by more or less diffuse and in some places a nodular type of tubercular inflammatory tissue. The tubercles show a striking tendency to proliferation of the epithelioid cells and the production of abundant fibrous tissue. In some areas, considerable masses of caseation may be seen, but in all places distinct multinucleated giant cells are characteristic. The hyperplastic reaction is predominating. Often miliary and conglomerate tubercles are seen, but in other places nothing but masses of epithelioid and giant cells, intermingled with areas of caseation are seen.

COMMENT

This child apparently did not appear as sick as he really was for no one realized that he would die so soon. This case shows marked lymphatic involvement in which the lungs showed nothing of great significance, which could not be explained by secondary dissemination. In children the point of inoculation is in the intestines, and also in the bronchial glands. In this case it was no doubt caused by lymphatic drainage of the tuberculous enteritis. Extensive involvement of the lymph glands in children is unusual. It is also unusual to find such a marked hyperplastic involvement without much caseation. This is why the glands appeared discrete and simulated Hodgkin's disease.

Literature reveals few cases such as the above. Most of the reports of enlarged lymph glands are confusing because of the lack of knowledge of the etiology of the cases. Since 1832, when Hodgkin described the glands known by his name, the relation between tuberculosis and Hodgkin's disease has been questioned. Many cases diagnosed as Hodgkin's disease clinically, proved to be tubercular in nature. C. Hilton Fagge[1] and Pye-Smith[2] reported such cases in 1873, as have Delafield[3] and Sternberg. Andrews[4] thought that although tuberculosis of the lymph nodes and Hodgkin's disease are indistinguishable clinically, the latter is a definite, saparate entity.

At times no visceral tuberculosis can be found clinically, and very few such cases have been described. Tedenat[5] in 1901 reported a case of a 18 year old male who presented enlarged maxillary, axillary and inguinal lymph nodes. No evidence of pulmonary or intestinal tuberculosis was found. Section of the gland was found to be that of tuberculosis. He reports another similar case in a man 40 years of age. Criep and Narr[6] reported a case of generalized tuberculous adenitis in a 19 year old white male, who was still alive at the time of the report. Section and inoculation of the epitrochlear glands showed tuberculosis. Most cases reported failed to come to autopsy where some old, if

not recent visceral tuberculous infection may have been found. Patton[7] reports a case, in 1914, which started with enlargement of the glands, and ran an acute course. At autopsy tuberculosis of the lymph nodes as well as of the viscera were found.

Landon[8] reports a case of tuberculous lymphadenitis, which clinically was mistaken for acute lymphatic leukemia because of a high lymphocyte count. This is a rare condition.

In this case, the mesenteric lymphadenitis was no doubt caused by the lymphatic drainage of the tuberculous enteritis, with spread to the other organs. Older writers commonly spoke of cryptogenic tuberculosis, such as commonly seen in cervical adenitis, without a demonstrable focus. Peribronchial adenitis, which is commonly seen, may be caused by the tubercle bacillus carried there by a phagocyte. Such explanation for regional tuberculous adenitis is not probable for a generalized infection, in which a lymphatic or blood dissemination is necessary. The cause for this type of tuberculosis may be the selective specific affinity of the tubercle bacillus for lymph nodes. This predilection of tubercle bacilli for lymph nodes is apparently not in harmony with Bartel's theory that lymph nodes are protective, or with Murphy's belief that the lymph cells act as inhibitory agents.[9]

CONCLUSION

The case reported is an unusual type of tubercular reaction, there being a lymphatic type of tuberculosis with all lymphatic structures being particularly affected. It is associated with an unusual degree of ulceration of the intestinal tract, affecting the lymphoid Peyer's patches. The origin is most likely from the intestinal tract, with invasion and spread by the lymphatics.

Clinically the condition was confused with Hodgkin's disease. Histologic and bacteriologic studies usually settle the diagnosis.

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—B—

Acute Infective Laryngo-Trachitis (Non-membraneous Croup)

LAVERNE B. SPAKE, M.D.
Instructor Ear, Nose and Throat.

December 26, 1929, we were called to see baby J., eighteen months old, who had been sick for the past three days with a cold, cough and hoarseness; as evening comes on, respiration becomes more embarrassed, with laryngeal spasm, difficulty in breathing, increased respiratory rate, stridor, restlessness, slight cyanosis of finger tips. During paroxysm there was a marked indrawing of suprasternal notch, and epigastrium; no temperature, a typical case of croup. Chest findings were negative, except a few moist rales over both lungs

December 28, 1929: All above symptoms were more intensified. Paroxysms were more frequent. Enlarged thymus was suspected, due to prominence of infant's chest, and the marked overweight. The baby was referred to Dr. Lewis G. Allen for *x*-ray of the chest.

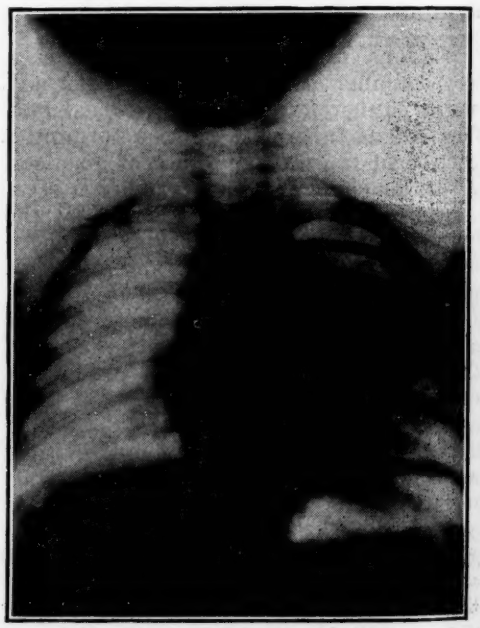


Fig. I

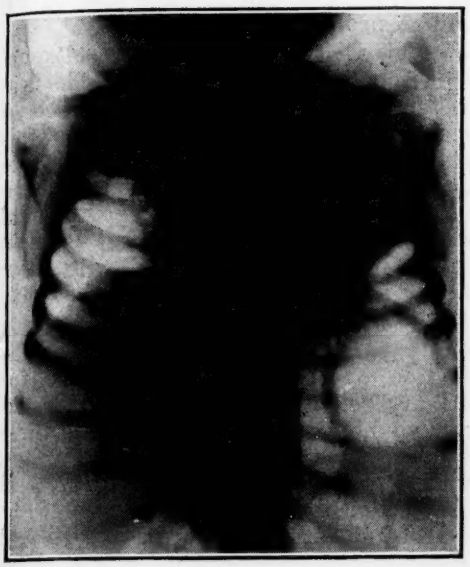


Fig. II

Plate I and II—December 28, 1929: "Radiographs of the chest show a marked increase in density of the entire left lung field, the increase is of a homogeneous character with no central parenchyma organization. No opaque foreign body is identified by repeated projections. The translucent shadow of the trachea is abnormally narrowed at the level of the 5th cervical vertebra.

Fluoroscopic examination of the chest reveals a wide excursion of each diaphragm. Expiratory excursion is exaggerated, to the end that the left chest appears quite opaque at this phase of respiratory cycle.

Some compensatory emphysema of the right lung field is noticed. No displacement of the heart has occurred.

The entire picture is rather classical of bronchial obstruction. No impression as to the type of obstruction is obtained by radio-graphic examination."

Physical chest findings: Limited expansion of left chest, decreased vocal fremitus, impaired percussion note, diminished intensity of the breath sound over the left chest. White blood count 21,000. Urine analysis, negative, no temperature.

Laryngoscopy was performed at Bell Memorial Hospital. Direct examination revealed a highly congested larynx, congestion extending over the vocal cords into the trachea, some tracheal edema. During the examination, the patient coughed up a dram or more of thick tenacious pus from the left bronchus. Bronchoscopy was not performed, because of acute inflammation of larynx and trachea, until further *x*-ray study had been made.

December 30, 1929, *x*-ray report showed "the lung field of normal transparency, diaphragmatic excursion is equal to the average and other changes reported at previous examination are not observed."

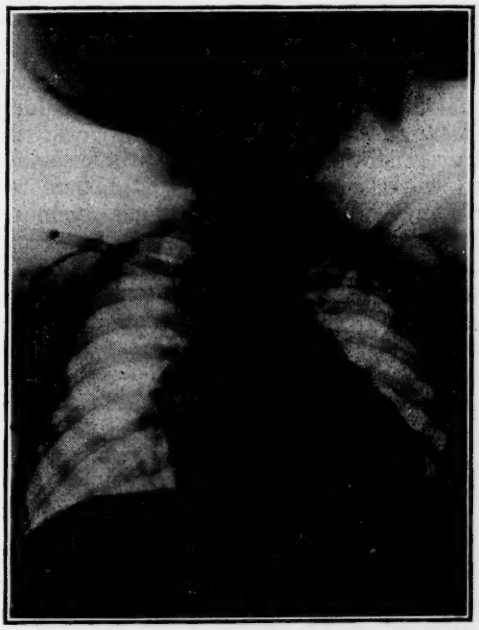


Fig. III

The third *x*-ray plate shows an entirely different left lung, which had been entirely blocked by the thick tenacious secretions from the larynx and trachea.

The patient left the hospital at the end of forty-eight hours, with no recurrence of symptoms, and no temperature.

SUMMARY

This type of case certainly indicates the wider field of usefulness of the laryngoscope and bronchoscopy; not only as a diagnostic instrument, but as a therapeutic measure in bronchial obstruction whether due to foreign bodies, chronic

bronchitis, bronchiolectasis or pulmonary abscess. The bronchoscope is used to examine the bronchi, to dilate constricted areas, for aspiration of accumulation of suppurative material in bronchi, irrigation of bronchi, instillation of medicine, or removal of specimen for biopsy, and in the diagnosis and treatment of certain types of asthma. Sweet of Cornell says that in lobar and bronchial pneumonia the pneumatic crisis can be promptly precipitated by bronchoscopic aspiration.

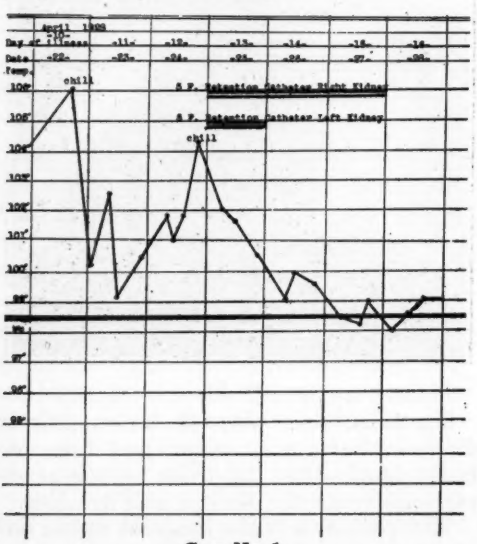
———R———

The Indwelling Ureteral Catheter In Renal Infection

CLINTON K. SMITH, M.D.

Department of Urologic Surgery

It has long been a well recognized fact that retention of urine in the bladder fosters the development of cystitis. Furthermore, infectious cystitis associated with retention shows stubborn resistance unless the treatment includes proper bladder drainage.



Case No. 1

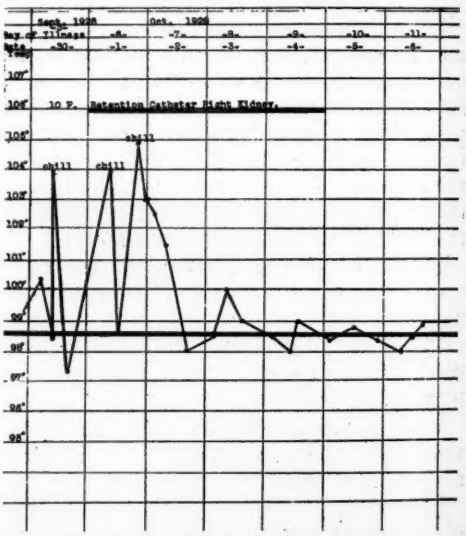
Female child, age 18 months. Chills, fever 101-106 ten days duration. Urine loaded with pus and coccoid bacteria. Leucocytosis 35,000. Prostrated. No 7F. catheters inserted and retained in each kidney. Double horizontal parallel lines on temperature chart denote period during which catheters were retained. Note steady decline of temperature. Uneventful recovery. Acute bilateral pyelonephritis.

Except in rare instances, the indwelling urethral catheter offers a practical and efficient solution of the bladder retention problem in these cases.

In view of the favorable results obtained with the indwelling bladder catheter, it is only a logical sequence of reasoning that we should turn to this plan in dealing with infection of the urinary tract above the bladder, particularly when clinical and experimental studies have convinced us that in most instances we are confronted with identically the same problem.

It is neither desirable nor essential, in this paper, to enter into a technical discussion of the factors responsible for urinary retention above the bladder, except to mention that these fall into three groups, namely, strictures of the ureter, faulty ureteral innervation, and ureteral obstruction incident to pregnancy.

It is further patent to the experienced observer that one or more of the above factors is the underlying problem in



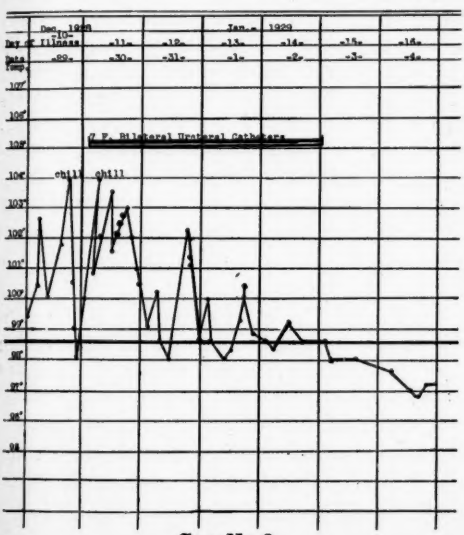
Case No. 2

Female age 26 years. Eleven days post partum, and ten days prior to examination this patient began to complain of chills, fever and frequency. Leucocytosis 19,000. Slight pain region right kidney. Urine loaded pus and colon bacilli, 30 c.c. retention kidney pelvis. Horizontal parallel lines indicate time period retention catheter. Note septic temperature curve, abrupt decline. Pyelitis of pregnancy. Post partum exacerbation.

most of the cases of upper urinary tract infection, usually designated as pyelitis, pyelonephritis and pyonephrosis.

The fact that a certain number of cases of upper urinary tract infection recover with the expectant, or internal medication, plan of treatment does not disturb the above viewpoint. In those cases wherein early recovery occurs we are, to all intents and purposes, dealing with a lesser degree of obstruction, and in the other instances where recovery is delayed—and which include by far the greater number—experience with the ureteral retention catheter is convincing that this plan of treatment not only materially shortens the course of the disease, but also reduces the hazard of more or less permanent renal damage from so-called back pressure.

There are two apparent objections to this plan of treatment. First, it may be contended that it is a technical procedure which can be carried out only by the



Case No. 3

Female age 48 years. Chills, temperature 101-104, ten days duration, following influenza. Patient prostrated, semi-comatose, tongue dry, uremic. Leucocytosis 13,000. Slight pain and tenderness region kidney. No. 7 F F. catheter inserted and retained in each kidney. Urine contained pus and coccoid bacteria. Residual urine 10 c.c. each k idney. Horizontal parallel lines indicate time period, retention catheters. Note steady, progressive decline of septic temperature curve. Mental condition cleared. Normal convalescence. Acute pyelonephritis following influenza.

cystoscopist. While this cannot be denied, it does not seem a valid objection in view of the benefits obtained in cases which do not respond readily to the internal medication plan, or in instances where the clinical situation is critical, with chills and high temperature. There is no contraindication to the introduction of a catheter into an acutely inflamed renal pelvis, providing it is not withdrawn leaving the already inflamed kidney to combat the development or aggravation of ureteral edema, which is probably already present and which is increased by the passing of the catheter.

Second, sentimental objection may be raised on the grounds that this plan of treatment is a distressing and dangerous procedure. Personal experience in a wide variety of cases is convincing that the retained ureteral catheter in calibre up to 16 French is well tolerated over periods of five to ten days and that the distress incident to the presence of the catheter is more than counterbalanced by the relief from fever, chills or renal pain. And further, that although the ages of these patients have ranged from eighteen months to sixty-five years, in no instance were there any ill effects noted. The appended brief case histories with accompanying temperature charts showing the results of the indwelling ureteral catheter were selected as representative of the several types of cases in which this procedure has proven its usefulness.

Richter's Hernia With Intestinal Obstruction

THOMAS G. ORR, M.D.

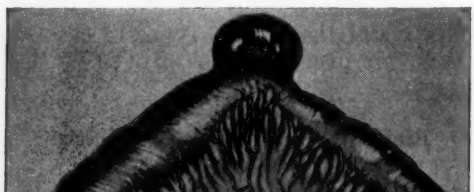
Department of Surgery

In 1778 Augustus Gottlieb Richter described a type of hernia which he called "the small rupture." It is characterized by a herniation or pinching of a portion of the circumference of the gut wall, which does not entirely obliterate the lumen. This type of hernia has been variously spoken of as partial enterocele, masked hernia, lateral pinching of intestine, lateral enterocele or nipped hernia, and sometimes Lavater's hernia.

It is said that partial enterocele was first observed by Fabricus Hildanus in 1598 and later by Lavater in 1672. Apparently Richter was the first to accurately describe the hernia, and hence it usually bears his name. Littré, in 1700,

mistook a hernia of Meckel's diverticulum for partial enterocele and the French now frequently speak of it as Littré's hernia. This error was not definitely corrected until more than a century later when Johann Meckel described the diverticulum.

The following very typical case of Richter's hernia is here reported: B. W., white female, age 65, was admitted to the Surgical Clinic of the University of Kansas, January 18, 1930. Her chief complaint was abdominal pain and vomiting. The present illness began 3½ days before admission, following the noon meal. She described her pain as "cramps in the pit of stomach, coming in waves." One hour after the onset of her pain she took a dose of calomel, which made her vomit. She has been vomiting since that time, whenever she has taken anything by mouth. There has been no fever. Her bowels have not moved since the onset of her trouble. Examination on admission showed slight abdominal distention with occasional visible peristaltic waves. Gurgling sounds could be heard on auscultation. There was no evidence of hernia or any abdominal mass. *x*-Ray of



Drawing of Richter's Hernia described in this case.

the colon with barium enema was negative for any obstruction. Urinalysis and blood counts were both normal. Blood chemistry showed chlorides 390 mgs. per 100 c.c., urea nitrogen 44 mgs. per 100 c.c., and CO_2 combining power of 43.8 volumes per cent. Soon after her admission her stomach was washed out and normal saline started by hypodermoclysis. She appeared improved very much within the next two hours, and the pain in her abdomen ceased. From the hour of admission, which was 4:00 p.m., until the following noon, she received 7,000 c.c. of normal saline solution. During the entire time she did not vomit, and distention, which was only slight, did not increase. About 1:00 p.m. on the following day she began to complain of recurrence of severe pain and vomited. It seemed evident that she was growing worse, and a diagnosis of complete intestinal obstruction was made. She was immediately taken to the operating room for an enterostomy. A right rectus incision was made under local anesthetic. Bloody fluid was found in the abdomen which suggested the possibility of strangulation of the gut. Because of the possibility of strangulation, it was thought best to explore the abdomen. The local anesthetic was then supplemented by gas oxygen and ether and the wound enlarged. The intestine was much distended and there was a loop which appeared to be fixed in the pelvis. In attempting to free this loop, the gut was torn and the peritoneum soiled with intestinal content. Through the torn opening a tube was placed and an enterostomy completed by the Witzel method. A drain was placed deep in the pelvis and the wound closed.

Following the operation she grew rapidly worse. There was no drainage from the enterostomy. Death resulted 13 hours later.

An autopsy was obtained and a partial enterocele or Richter's hernia of the lower ileum was found fixed in the right femoral canal.

One of the best discussions of Richter's hernia is that of Sir Fredrick Treves[1] published in 1887. He collected 53 cases and noted that the condition is more common in women in the fifth decade of life, and that the majority occur as femoral hernias on the right side, involving usually the lower ileum. He further noted that the partial enterocele occurred more frequently in old than new femoral hernias. In these cases collected by Treves the operative mortality was 55 per cent and the non-operative 75 per cent. Spontaneous cure may result with

the formation of a fecal fistula. Of the 53 cases 38 were femoral, 13 inguinal and 2 ventral.

It has been noted that when ⅔ or more of the circumference of the gut is involved in the strangulated portion, water will not pass through because of the acute angulation. In this type of hernia gaseous distention is not usually marked. The constriction may be water tight and not air tight, and hence the possibility of gas passing through the angulation. Low[2] noted that vomiting was less frequent, less severe, often of late onset and rarely feculent. In general the symptoms are less severe than in other types of bowel strangulation. The bowels may be open and even a persistent diarrhoea has been known to exist. He reported 4 cases, all of which were women with right sided femoral hernias. Pain is usually referred to the epigastrium or umbilical region and is of little value in localizing the hernia.

SUMMARY

It is interesting to note how nearly the findings in the reported case conform to the rule. The patient was a woman, past 50 years of age, with a right sided femoral hernia, not evident on examination and without a history of hernia. The lower ileum was involved. The diagnosis was not made during life. Vomiting and distention were less marked than in the usual obstruction of the ileum. The location of the pain, which was above and to the left of the umbilicus, did not aid in locating the lesion.

An observation was made in this case that may be made in other cases of acute intestinal obstruction in which large quantities of sodium chloride solution are given. The vomiting ceased for nearly 24 hours after the treatment. This evidence of general improvement may lead one to a false conclusion in regard to the patient's condition, and cause harmful postponement of operation.

Although Richter's hernia, causing strangulation and obstruction of the small intestine is quite unusual, the condition is sufficiently common to be kept in mind when symptoms of acute intestinal obstruction develop in a woman past fifty years of age in whom the cause for the obstruction is not evident. A careful search should be made for a small femoral hernia in all suspected cases.

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Three Cases of Tularemia

CORA M. DOWNS, Ph.D.; A. M. FORTNEY, M.D.; and E. R. EDMISTON, M.D.

Department of Bacteriology and Immunology, University of Kansas

Since the first case of *B. tularense* infection in man was reported in 1907[1] there have been an increasing number of cases recorded in the literature. Francis adequately reviewed the literature on tularemia in 1925[2] and again in 1928.[3] One case in Kansas was reported by Gillette in 1927[4] and since that time eight cases have been reported to the Kansas State Board of Health.[5] We have been able to find record of 1,329 cases reported since 1928. Many of these occurred in a large epidemic in Russia in 1928.[6] Although prior to 1927 tularemia was supposed to be a disease which occurred only in North America and Japan, laboratory infections have occurred in England and recently there have been some cases in South America. It is difficult to say whether the disease is becoming more common or whether it is only more frequently reported. It is well known that infections caused by organisms belonging to the hemorrhagic septicemia group tend to increase in frequency over a number of years and then decrease spontaneously. This is well exemplified in the occurrence of epidemics of bubonic plague and it might very well be since *B. tularense* is a closely related organism that the incidence of tularemia would resemble that of plague. In plague a reservoir of infection resides in rats and other rodents and epidemics in man are coincident with epizootics in animals. In tularemia the animal reservoir exists in the wild rabbit and other closely related rodents. The three cases here reported were confirmed serologically and *B. tularense* was isolated from one of the cases. All of the cases were clearly infections from

wild rabbits, the common wild cotton tail of Eastern Kansas.

Mr. and Mrs. I. were seen November 29, 1928. Mr. I had a draining sinus in the epitrochlear region. This had been preceded by swelling and pain in the epitrochlear glands which had suppurated and been drained. He had a healing sore on the second finger of his right hand and the axillary glands on his right side were red and swollen. Mrs. I. had a sore on her left thumb which was practically healed but there was a chain of hard and swollen lymph glands extending from the middle of the flexor surface of the forearm into the epitrochlear glands. The axillary lymph glands were palpable. The history of these two people is as follows: October 20, 1928, Mr. I. had dressed several wild cottontail rabbits which he had shot. One rabbit had been discarded because of spots on the liver. These rabbits were cooked by Mrs. I. Two or three days later both Mr. and Mrs. I came down with what they called "flu." They had fever, prostration and sweating and were ill for about ten days. At the same time they noticed soreness of the axillary glands and the beginning of a felon on Mr. I's. finger. Mrs. I. already had a sore on her thumb which no doubt served as a portal of entry for *B. tularense.* These so-called felons drained but did not heal well. They became alarmed because of the persistent swelling and soreness of the regional lymph glands. They then consulted a physician (AMF). On November 29, a small amount of pus was obtained from the draining epitrochlear gland in Mr. I's. arm. Pus was also aspirated from a swollen gland near the median basilic vein from Mrs. I's. arm. Blood from both was also obtained from the arm vein. This was sent to Washington to be tested for the possible presence of agglutinins for *B. tularense.*

The pus from both cases was examined in direct smears stained with carbolfuchsin and Wright's stain but only disintegrated pus cells could be seen. The pus was emulsified in saline and injected subcutaneously into guinea pigs and a rabbit. The animals survived several weeks. The blood from both cases was reported from the Hygienic Laboratory to show positive agglutination in a dilution of 1-1280. This with the history and symptoms might be considered to have established the diagnosis of tularemia. An agglutination test on both serums was made in our laboratory using *Br. melitensis* because of the cross agglutination which is reported between this organism and *B. tularense.* Mrs. I's. serum agglutinated *Br. melitensis* at 1-80 and Mr. I's. was doubtfully positive at 1-10. December 5th, the patients were again seen. Mrs. I's. glands were smaller and less painful. One of the axillary glands in Mr. I. was ready to lance. This was done and about 15 c.c. of pus was evacuated. This material was ground in a sterile mortar, strained through gauze and inoculated into guinea pigs but with entirely negative results. Francis[2] reports that *B. tularense* can seldom be recovered by animal inoculation longer than one month after the onset of the disease. The material in these cases was first collected approximately six weeks after the onset of the infection, and this no doubt accounts for the negative inoculation results. Francis reports that the organism has been cultured only once directly from human lesions though it can be cultivated from the blood and organs of infected animals rather easily by using the proper media. The organisms survive for as long as three weeks in pus mixed with an equal part of glycerine and for six weeks in spleen from an infected animal when the spleen is immersed in glycerine. The diagnosis of tularemia was based on the history of dressing rabbits, the local lesion on the fingers and the acute febrile attack accompanied by persistent lymphadenitis of the regional glands together with the serological findings.

The third case is that of a young negro, aged twenty-six, first seen January 27, 1929. He had been hunting January 5, and had cut the index finger of his left hand on a sliver of bone while dressing a rabbit. He became ill January 9, and soon after noticed that the cut on his finger did not heal. He was ill and in bed, complained of fever, lack of appetite and prostration. On January 27,

his blood count was as follows: Hemoglobin 70-75 per cent; red cells, 3,440,000; white cells, 3,800; neutrophilic leucocytes 57 per cent; small lymphocytes 35 per cent; mononuclears 5 per cent; eosinophiles 1 per cent; basophiles 2 per cent; normoblasts 2 per cent; poikilocytosis and aniscocytosis and polychromatophilia were marked.

A sample of blood was sent to the Kansas State Board of Health Laboratory and to the Hygienic Laboratory at Washington to use for an agglutination test with *B. tularense.* Both reported positive for *B. tularense,* the latter in a dilution of 1-1280. Wassermann and Kahn tests were made because of the anemia shown by the patient but both were negative. The agglutination test with *Br. melitensis* and *Br. abortus* was negative. A swab taken from the draining sore on the finger was rubbed on the plucked abdomen of a guinea pig. The pig died two days later with gray swollen lymph glands in the inguinal region, spotted liver and spleen. The appearance of the liver and spleen is very characteristic in infections with *B. tularense.* They are thickly covered with small grayish white spots which on section are found to be areas of focal necrosis. Two guinea pigs and three rabbits inoculated with material from the spleen, lymph glands and liver of the original pig, all died with typical lesions within one week after inoculation. The organism was cultivated from the hearts blood of one of the inoculated guinea pigs and has since been maintained on cystine dextrose blood agar. This organism causes the death of a guinea pig in two or three days when injected subcutaneously in very small doses. It will be noted that this material was collected from the patient about three weeks after the infection took place. An agglutination test made with the serum collected from the patient January 27, was set up against the following strains: the strain cultivated from the patient, known as AT 29; a supposedly avirulent strain from Dr. Shaw at the University of Virginia, BT 29 and one received by the Kansas State Board of Health from Dr. Francis, CT 29. His serum agglutinated all these strains in dilutions of 1-500. BT 29 and CT 29 were tested for virulence by the inoculation of 5 c.c. of a suspension from a slant culture. The pigs receiving CT 29 died in eight days with typical lesions. The pigs receiving BT 29 survived, showing that CT 29 is not entirely avirulent. The patient was again seen March 10, 1929. He had gained about ten pounds and complained now only of a slight weakness and pain in the chest over sternum and in the region of the spleen. The sore on his finger was entirely healed. The glands in the epitrochlear region on his left arm and a chain extending up into the axilla were hard and about the size of a lima bean but not tender. There was a gland in the axilla which was hard, somewhat tender and about the size of a small hen's egg. The spleen was somewhat enlarged and tender. His blood count at this time was as follows: Red cells, 4,052,000; hemoglobin, 80 per cent; white cells, 9,600; neutrophiles, 66.5 per cent; small lymphs, 25.5 per cent; mononuclears, 5.5 per cent; eosinophiles, 1,5 per cent; basophiles, 0.5 per cent. There were a few poikilocytes and several normoblasts. The agglutination tests with AT 29 gave a good positive at 1-250 and slight positive at 1-500. The organism isolated from this case is still virulent for guinea pigs and rabbits even when applied to the uninjured skin.

There are several points of interest in this second case. So far as we can ascertain no anemia has been reported in connection with tularemia, although from the protracted course of the disease an anemia might be expected. Usually a leucocytosis of moderate extent is reported but in this case there was a leucopenia. An attempt was made to account for the blood picture on some other grounds but without success. The physical findings and history seemed to show no evidence of any disease other than the present infection with *B. tularense.*

An attempt was made to ascertain if anyone had noticed an epidemic disease among the wild rabbits in this vicinity but apparently nothing of the kind had been noticed. The disease is probably

endemic among the rabbits of eastern Kansas.

Three cases of tularemia of the ulceroglandular type with typical serological findings are reported. *B. tularense* could not be isolated from the first cases probably because of the length of time which had elapsed between the time the infection took place and the time the culture was taken. A typical strain of *B. tularense* was isolated from the third case. Francis has pointed out the similarity between tularemia and sporotrichosis. This similarity was very evident in the first two cases here reported. Tularemia has also been confused with tuberculosis and microscopically both human and animal lesions closely resemble tubercles. The history of contact with rabbits or other rodents and the serological findings are important in making a diagnosis. Whenever possible animal inoculation should be done during the first three weeks of illness. Because of the fact that material from an infected animal spleen or in pus will keep well in glycerine it is always possible to send in such material to a laboratory for identification. The mortality in human cases is low (3 per cent) but the infection is extremely disabling and long drawn out. The first two cases still had draining glands in January, 1929, and Mrs. I's. axillary glands were still draining in January, 1930. The third case had glandular swelling and complained of weakness two months after infection.

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Some of the Important Signs and Symptoms In the Diagnosis of Brain Abscess

O. Jason Dixon, M.D.

In a recent lecture by Robert Lund on "The Diagnosis and Differential Diagnosis of Otogenous Brain Abscess," he states that the chief reason of the high mortality in brain abscess is the fact that of all the otogenous complications brain abscess most frequently escapes being recognized in due time. In a total of fifty-four patients with brain abscess, from the Otological Department of the Municipal Hospital in Copenhagen, which Lund studied and reported in detail, only twenty-nine were diagnosed and evacuated (51 per cent); nineteen were not diagnosed (34 per cent); six were diagnosed, but not evacuated (11 per cent). Of the twenty-nine patients diagnosed and operated, only ten recovered (18 per cent of the total number of cases). He considers this material a universal expression of what is being done for brain abscess.

It is obvious that the chief factor of importance in the management of brain abscess is the diagnosis. As I see it, the obstacles to overcome in the diagnosis of brain abscess are first, that it is a problem which may confront any physician in any field of medicine; second, that the signs and symptoms are about as clear cut as any other lesion in the body; third, that a thorough knowledge of neurological anatomy may be of considerable help in the diagnosis of brain abscess, but is by no means essential. Absence of knowledge of neurological anatomy does not excuse a failure to diagnose brain abscess, but it is probably the most frequent reason for the failure to recognize this lesion.

The ideal condition would be for the neurologist, or brain surgeon, to have in his care a brain abscess patient from the onset of the primary symptoms. This, of course, seldom happens, and, therefore, it behooves all of us to know something about the early signs and symptoms of brain abscess.

ETIOLOGY

Korner first expressed the principal rule that otogenous brain abscess is practically always localized in the proximity of the suppurating middle ear and mastoid process, *i.e* the temporal lobe or cerebellar hemisphere on the same side. He has also shown that eighty-five per cent of all brain abscesses are otogeneous in origin. v. Bergman insists upon the claim that the diagnosis of brain abscess can be made only where a possible primary focus is demonstrated. Lund has shown that fifty per cent of all otogeneous brain abscesses are due to a

cholesteatomatous middle ear lesion. Therefore, with these well substantiated facts, it is of the utmost importance that we try to determine the age and location of the primary focus, and its subsequent history. It is quite obvious that the above facts can be determined by any physician, without the aid of very much special mechanical diagnostic equipment.

Dandy has recently brought out the advantages of late surgical interference in the treatment of brain abscess. I heartily agree with his opinion in the matter. However, late surgical interference does not imply that there is advantage to be gained in a late diagnosis. It has been demonstrated that a fully developed brain abscess may be of only fourteen days duration, and there is one reported case of an otogenous brain abscess of sixty-eight years standing. It should also be borne in mind that a fully developed brain abscess, that has been latent for years, may suddenly manifest most violent and fatal symptoms. Trauma often plays a part in such cases. Generally, however, the latent period may be said to last from two to six weeks.

DIAGNOSTIC SYMPTOMS

Diagnostic symptoms may be divided into initial, latent, manifest and terminal. These may all vary, and usually overlap. The patient is usually diagnosed in the manifest or terminal stage, at which time he has two classes of symptoms, which may be divided into general or systemic and local or brain symptoms. Under general we have a marked lethargy, sallow complexion, emaciation, loss of appetite, constipation and subnormal fluctuations of temperature of about two degrees. The most characteristic finding in the history of the brain abscess patient is the negative stage, *i.e.*, he has no complaints, except one—headache—which falls under general brain symptoms, and, unless he is in a late, or terminal, stage, headache is the most constant and most severe symptom. This headache, which a brain abscess patient complains of, is unlike any other type of headache, except possibly that of brain tumor. It is unrelieved by any form of medication, it is constant; gets progressively worse, and, were it not for the lethargic, toxic state into which the patient rapidly sinks, it would no doubt be the means of rescuing him from his disastrous condition more frequently than any other symptom. Other general brain symptoms which are usually present are vertigo, sudden vomiting, slow cerebration, bradycardia, photophobia, and last, and of most infrequent occurrence, optic neuritis.

Since this paper does not deal with the localization of brain abscess, that part will not be considered. However, cranial nerve lesions do play an important diagnostic part, and, particularly is this true, in lesions of the third, sixth and seventh cranial nerves of uncertain etiology

SEROLOGY

The patient with a brain abscess usually has about 12,000 leukocytes, and a moderate decrease in the red blood cells and hemoglobin. As the disease progresses. The spinal fluid cytology varies with the different stages of the abscess. As I have already discussed this subject under a separate paper, I shall only mention the chief diagnostic aid in the study of spinal fluid *i.e.*, whether the patient has a so-called sterile, or a septic meningitis. The pressure readings of the spinal fluid, and the reaction of the patient following these lumbar taps, is, in some cases, of striking importance. It should be borne in mind that in every patient with a brain abscess, the primary stage is closely associated with a diffuse meningitis, and the local meningitis, particularly in abscesses of otogenous origin, may, at the onset, present such a confusing picture as to make differential diagnosis impossible.

SUMMARY

The following tables were made up from a small series (twenty-five) of brain abscess patients that have come under my observation. The symptoms and signs were taken from recorded observations and histories that were made by the interns, the nurses and myself. These tables are in themselves a summary of the various signs and symptoms which these patients with brain abscess had.

AGE INCIDENCE:
- 10-20 years 9
- 20-30 " 4
- 30-40 " 6
- 40-50 " 1
- 50-60 " 3
- 60-70 " 1
- Unstated 1

SEX:
- Male 15
- Female 10

PREVIOUS MASTOIDECTOMY:
- Following mastoidectomy 14
- No mastoidectomy 11

CHOLESTEATOMATOUS INVOLVEMENT:
- Cholesteatomas 4

RESULT:
- Recovered 6
- Died 19
- Autopsies 9

PRIMARY FOCUS:
- Draining ears 14
- Eyes 1
- Infection of antrum from tooth extraction.. 1
- Influenza 1
- Lungs 2
- Osteomyelitis of hip with dural involvement 1
- Frontal Sinus 3
- Unstated 2

GENERAL SYMPTOMS:
- Choking
- Convulsions
- Delirium
- Difficulty in swallowing
- Diplopia
- Disorientation
- Dizziness
- Draining ears
- Drowsiness
- Earache
- Epistaxis
- Headaches
- Holding of head to side
- Instability
- Motor Symptoms
- Numbness of face, mouth, tongue, extremities
- Paralysis
- Rigidity of neck
- Slowness in response
- Vertigo

FORMS OF HEADACHE:
- Frontal 6
- Frontal and occipital 1
- Frontal and parietal 2
- General 4
- Occipital 2
- Unstated as to type 1
- Not recorded 9

FORMS OF PARALYSIS:
- Facial 5
- Facial and Side 5
- Side 4
- Partial 1
- None 10

	Not Diagnosed	Diagnosed Not Evacuated	Diagnosed and Evacuated	Total
Cerebellar	....	....	8	8
Abscess left temp. lobe	....	....	2	2
Abscess right temp. lobe	....	1	3	4
Abscess multiple	....	1	....	1
Abscesses dural	....	2	1	3
Unstated	1	2	4	7

Scientific Jackasses

PARKE WOODARD, M.A., M.D.
Assistant Professor of Physiology

Not so long ago an old doctor said in my hearing that he had advised his son, who wished to study medicine, against going to one of our leading medical colleges because this institution made "scientific jackasses" of its students. On further questioning he explained that people entered medical school to learn how to treat the sick but that while there they spent their time manipulating mechanical trinkets such as gas analysis and elecrocardiographic apparatus, occupations of quite doubtful value for most doctors in view of the fact that not many of them would have access to such instruments later on in practice. This old doctor is only one of many who are of the opinion that now-a-days we are trying to make the study of medicine too scientific and that instead of training students to use their own sensès in the detection and treament of disease we are telling them to rely on machines, solutions and test tubes to make diagnoses and do their thinking for them. Even our first and second year medical students are of the notion that they devote too much time to stimulating frog legs and producing colored precipitates in test tubes, and have decided to have done with such nonsense in the easiest way possible in order to go on to the clinical subjects where "real medicine" is learned.

Perhaps some of the difficulty lies in a failure to appreciate what scientific medicine really is. To most people a scientist is one who produces reactions in test tubes, takes readings from dials and indicators and in general works with apparatus or makes inanimate material behave properly. The idea of subjecting a variable organism like a sick person to the working of an invariable machine or chemical reaction for the purpose of diagnosis is repugnant. In some way science when concerned with medicine has come to mean laboratories and apparatus entirely divorced from diseased individuals, and the "scientific physician" is regarded as one who more or less cold bloodedly looks into his micro-

scope and test tubes without much thought of the variable human being he treats. If his apparatus be taken away from him he is lost and ineffective.

If the above is "scientific medicine" then of course the less of it we have the better, but there are some of us who hold different ideas. To be scientific is to be willing first to carefully collect all the information possible pertaining to a problem; and secondly to critically evaluate and interpret such data. In the practice of medicine the problem is presented by the sick individual and exactly the same careful observation and critical judgment should be brought to bear in dealing with it that the chemist uses with his solutions and test tubes. Just because the physician is confronted by processes many times more complex is no reason why he should be satisfied to found his conclusions on insufficient evidence and let slipshod and inaccurate methods satisfy him. The history of the advances in medicine has shown that it is only by carefully checking up that the wheat has been separated from the chaff in proposed methods of diagnosis and treatment. Accurate observation combined with critical evaluation has more than once been the key to the solution of a problem, and such procedure is a good habit for medical students to follow.

A laboratory is one of the best places to practice accurate observation. To be sure the student here sets up artificial conditions which are seldom met with in actual life but that is of advantage. By controlling all the factors involved the effect of variations in any situation may be better appreciated. The investigator has the opportunity to look in on life processes, see them at work and then by changing one or two of the ingredients to note the effect. Only by using the laboratory method can he be sure that the factor he changes produces the result he obtains—because only by this method can he keep all the other factors constant. Thus the very valuable habit of buttressing conclusions by controlled experiments is illustrated and emphasized. If this idea alone were wide spread it would save the printing every year of thousands of pages of medical literature which at present are of no value because the authors have not taken the trouble to control their results.

Finally, a perusal of medical literature shows that a large percentage of the advances in medicine have come from the laboratories. A list of these would certainly include the discovery of insulin; the development of the liver diet in anemia; the understanding of many obscure points in nutrition; and many more. These advances are used every day now by even the most obscure practitioner but to reach this position has taken years of careful, well controlled experimentation; very often involving test tubes and solutions and sometimes involving stimulation of frog legs. A former teacher of mine in medical school told me that formerly he made sport of playing with white rats with the idea of gaining any medical truth; but since the war he didn't do so. Then he was in charge of some nutrition work for the Red Cross in the Balkan States. He was surprised to learn that he could make direct use of information which had been collected from feeding experiments on white rats. Since then he has been very loath to say that even the most useless appearing scientific work is without value.

Repair of Complete Perineal Laceration

RALPH R. WILSON, M.D.

Department of Gynecology and Obstetrics

The lesions commonly known as third degree lacerations or complete rupture of the perineum are, to paraphrase it, like "the poor, ye shall have them with ye always." It is their frequency that has prompted this effort.

There is no claim for originality in this discussion nor is any effort made to condemn any method that has functional results as the end-objective. There have been many techniques devised since the original was offered by Thomas Emmett, who is considered the father of modern perineorrhaphy. To those who have satisfactory techniques there is nothing to be said, but there are some who are not satisfied. This operation can readily be combined with other vaginal procedures

as trachelorraphy, removal of Bartholin cyst or hemorrhoidectomy. It may even be combined with laparotomy. That I say because I am of the opinion that true cystoceles are best treated by some form of uterine suspension in addition to the vaginal work. In dealing with these cases, however, one is always impressed with the rarity of cases exhibiting co-existing hemorrhoids or uterine prolapse with complete rupture of the perineum.

The practitioner who does obstetrics, whether he be a specialist or not, is directly responsible for complete laceration in one of several ways:

First, through faulty management of labor the lesion may be produced.

Second, even though the lesion be entirely unavoidable, which is quite possible, he is equally at fault if a satisfactory repair is not done.

Third, occasionally cases of pregnancy have old complete tears, and the man is not very thorough who does not do a repair at the time of subsequent confinement. It is to be kept in mind, however, that herniations through the pouch of Douglas may be present, and of course are usually not to be attacked at such times. Furthermore, those cases possessing contraindications to general anesthesia had better be postponed and handled under special anesthesia.

As a matter of fact, modern obstetrics is really a well-developed field in specialized major surgery, and can no longer be considered mere expectant midwifery.

Personally, I am very much in favor of doing these repairs, whether on old or new tears, as soon after delivery of the child as possible, because:

1. The patient is saved the trouble and expense of entering the hospital later.

2. Fresh lacerations should be repaired before infection develops.

3. It is now well known that the tissues of a recently delivered woman heal very satisfactorily.

4. If the proper technique is used the lochia is not a contraindication at all.

5. Old lacerations should be dissected up and repaired, because labor should be quite easy in a multipara with no perineum; and therefore, she can stand prolongation of the anesthetic. Furthermore, because of the very nature of the tear any mechanical assistance at delivery is easily accomplished.

6. These operations should always be followed with a rigid diet regime for a few days.

It is better, therefore, to have the mother on a diet at the time before the milk comes in, than later when the baby really needs it.

PREOPERATIVE PREPARATION

1. Digitalization of the heart in suspected or proved cases of cardiopathy is of great value.

2. Hospitalization is not entirely necessary but the nearer the conditions approach that of a hospital the easier it is for the operator both from the mental and physical standpoints.

3. The anesthetist should be chosen with as great care as if for a laparotomy.

4. If done immediately after delivery the rectum and lower bowel should be irrigated clean with soap solution.

5. Pituitrin, one cubic centimeter, is given intramuscularly in the left upper arm as soon as delivery is imminent, not to assist in expulsion at all, because the child is practically born, but to make the uterus clamp down on the placenta and prevent bleeding during the operation or perhaps expel it entirely by the time the cord is dressed and other preparation made.

6. Catheterization should always be done, whether in conjunction with obstetrical delivery or as a regular secondary repair, because the urethra pierces both layers of the triangular ligament, which is considerably handled during the operation, and frequently contributes to urine retention during the early convalescence.

Now as to post operative cases one seeks to do two things:

First. Secure comfort for the patient.

Second. Promote healing of the tissues.

For the average case we have adopted the following instructions to the nursing attendants:

1. Vaseline gauze to external suture line constantly for first twenty-four hours.

2. Give opiates freely.

a. Morphine hypodermically or morphine with lead acetate per rectum as often as needed to control gas pains or tenesmus.

b. Tincture of opium m.x.t.i.d. daily.

3. Post operative catheterization every 12 hours, if needed, until the edema of the parts is down, when the patient may be sat up erect.

4. Diet to be (RRW) liquids or without residue.

5. No milk, enemata nor catharsis.

Now as to the first enema, opinions (RKW) vary from the 4th to the 10th day. I recall one case with troublesome distention on whom we passed a rectal tube the second day without harmful results. The patient's condition is the best indicator up to the 10th day. But always the cleansing enema is preceded by an oil retention enema and if possible mineral oil is given in large doses by mouth every 6 hours for one day beforehand.

The average case when combined with delivery is discharged from the hospital not later than the 15th day.

Chorea In Children

HUGH L. DWYER, M.D.
Department of Pediatrics

Chorea is probably a manifestation of rheumatic fever, a symptom rather than a disease entity. It is a self limited condition and seldom is there an opportunity to study the anatomical changes at necropsy. In the few cases which have been studied the lesions consisted of perivascular cellular infiltrations in the region of the basal ganglia, the neostriatum and the corporus striatum similar to those found in epidemic encephalitis.

The disease is more frequent in early life, about 80 per cent of the cases occur during the period between 5 and 15 years. The symptoms of rheumatic fever in children differ somewhat from those in adults. In children it usually begins with a slight fever, throat infection and often a cervical adenitis. The duration of the fever and throat symptoms are irregular. They may be very slight and subside in a few days, to be followed in a week or two by endocarditis, chorea or both. On the other hand swollen painful joints, muscle pains occurring as leg-ache while in bed at night, the so-called growing pains, may precede the nervous or cardiac manifestions.

In 72 cases[1] that I have followed the seasonal incidence was marked, about 50 per cent occurred in the months of March, April and May. They were about equally divided as regards sex although the condition is said to be more common in girls.

Although the tonsils are regarded as the portal of entry of the infection, in 12 of the 72 patients in this series, the tonsils had been removed months or years before chorea appeared.

Recurrences are common, 12 patients had a return of the symptoms within a year following the first attack. Several had more than two attacks, although the tonsils and adenoids had been removed during the first hospitalization.

Cardiac complications, a mitral murmur in each instance was found in 20 of these patients (about 28 per cent).

DIAGNOSIS

The diagnosis as a rule is not difficult. Before the active symptoms are noticed, the parents will usually observe a clumsy attitude in the child especially at the dinner table. Over-reaching and dropping the eating utensils are commonly reported. In a few days facial grimaces and aimless movements in one arm and shoulder make their appearance, and these purposeless movements, more marked while the child is observed, soon spread to the other side.

TREATMENT

The general plan of treatment is to remove the child from the usual surroundings, preferably to the hospital, and provide absolute rest in a quiet, darkened room. He should be free from all external stimuli for about two weeks and with as few visitors as possible. Sedatives may be necessary during the worst stages and when this period is passed, the tonsils and adenoids if present should be removed and likewise den-

tal infection. The tonsils should be removed without waiting for the residual symptoms to disappear as these may persist for three months. The presence of a mitral murmur is in itself no contraindication to an anesthetic, provided the process is not acute, and signs of decompensation are not present.

We have tried a great many drugs and therapeutic procedures. Spinal puncture, the intraspinal administration of the patient's serum, horse serum, specific horse serum obtained after injecting the streptococci from chorea patients into horses, all have been used in this series but the results have not justified their continued use Neoarsephenamine was used in four cases without notable improvement.

Sodium bromide and hydrated chloral in suitable doses gave relief during the period of great restlessness. Sodium saliglate in large doses given per rectum in about one ounce of starch water, 60 to 120 grains, was used early in the disease in the few patients having fever or any active manifestations of rheumatism, such as joint pains or a recent cardiac involvement.

One of the most satisfactory sedatives used was magnesium sulphate in 25 per cent solution. It was given once or twice daily in 15 c.c. doses subcutaneously, and in some cases was given for three weeks.

Phenobarbital in doses of three-fourths of a grain, once or twice daily proved to be a very satisfactory sedative, when it is necessary to treat the child at home. I believe that the quiet room, absolute rest in bed, phenobarbital in the most violent stage and Fowler's solution of arsenic for the residual symptoms are the best methods of treatment. In the hospital the long period of very mild choreic symptoms seems to be benefitted by two or three intravenous injections of typhoid vaccine, 20 to 40 million organisms at a dose, repeated in four days. The febrile reaction that follows certainly brings about a cessation of symptoms, but this procedure should be reserved for the patient in the hospital.

After a trial of most of the drugs and therapeutic procedures that have been advised, we have discarded all except a few of the simple procedures, viz:

1. Rest in bed in a quiet darkened room for at least three weeks with no visitors, books or anything to hinder complete rest and relaxation.
2. For the control of symptoms during the acute stage, phenobarbital once or twice daily.
3. Warm tub bath when needed to induce relaxation and sleep.
4. Fowler's solution of arsenic after the most active symptoms disappear, for a period of two or three months.
5. Removal of tonsils and adenoids and dental infection as soon as the active symptoms subside.
5. The patient should not be kept in bed until all symptoms clear up. Those with the residual symptoms can be taken out in a wheel chair in fine weather, or allowed to walk about quietly when such effort is not tiring.

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Safeguards In Spinal Anesthesia

ANNA M. WENZEL, M.D.

Department of Surgery

During the last few years spinal anesthesia has become again exceedingly popular. The reasons for this renewed interest are newer methods, improvement in technic, controllable anesthesia, and the prophylactic use of ephedrin against vasomotor collapse. Because of its present wide usage relatively definite conclusions may be drawn, concerning its usefulness, reliability and dangers.

Sise[1] recently estimated that the death rate in Greater Boston, during a period of one year, amounted to 1 in 100 in spinal anesthesia. At the Lahey Clinic there occurred one death in 950 cases. Babcock's[2] average is one death in 1,000 cases. Rygh and Bessessen,[3] from a review of the literature, concluded that there was one death in 3,345 cases. The most important factors accounting for some of the high death rates at the present are; the inexperience of the anesthetists with this type of anesthesia, the poor selection of patients, lack of knowledge of physiology, overdosage, the slow, inadequate and indecisive treatment of patients during anesthesia, and to a certain

extent the more general use of this anesthetic for abdominal surgery.

The contraindications to spinal anesthesia are relative. Any patient whose vitality is at a low ebb, whether it be from long illness, sepsis, old age, shock, hemorrhage, or a moribund state is not a very suitable case for spinal anesthesia. In the extremely sick, when local anesthesia cannot be used, an inhalation anesthesia may sometimes be preferable to a spinal anesthesia. In the presence of central nervous system disease, especially of the spinal cord, most every one is of the opinion that spinal anesthesia should not be used. Low blood pressure per se is not a contraindication, since these cases are easily controlled with the proper use of ephedrin. Stout[4] believes that spinal anesthesia is limited only by the skill and experience of the anesthetist.

There exists a difference of opinion as to the limitations of spinal anesthesia in the presence of certain pathological states. Marked hypertension associated with an enlarged heart, gastrointestinal perforation, acute peritonitis, or localized perintonitis or abscess, and strangulated hernia and ruptured appendix are some of the contraindications that are enumerated by some authors[5, 6, 7]. Others disagree with these contraindications and recommend it in intestinal obstruction, gastrointestinal perforation, and suppurative conditions within the peritoneal cavity[4, 8, 9, 10]. Most workers feel that spinal anesthesia should not be used for surgery above the diaphragm.

The advantages of spinal anesthesia are; a marked relaxation, no metabolic disturbances, no bad effects on the lungs, kidneys, heart or other vital structures, less postoperative distress, and less danger of postoperative respiratory complications.

For any patient showing evidence of dehydration Sise recommends the use of 1500 c.c. of saline solution plus 50 gms. of glucose given hypodermically or intravenously just preceding the anesthetic. In his experience these patients with their vascular system well filled with fluid respond more effectively to treatment and are more resistant to a drop in pressure. Another factor of importance is sufficient preoperative medication to allay as much as possible all mental strain and worry, as the success of an anesthetic partially depends on the psychic calm. In the Surgical Clinic of the University of Kansas the average individual receives from 3 to 6 grs. of amytal by mouth 2 hours before the operation, and morphine 1/6 to ¼ gr. with scopolamine 1/300 to 1/150 gr. one hour later. The size of the dose depends upon the weight and the condition of the patient. In the debilitated, and especially in the aged, scopolamine is omitted and a small dose of atropine is substituted. The premedication in such patients is reduced to a minimum, so that they may not lie in stupor and inactivity for hours after operation. Another reason for giving amytal is to counteract the possibility of novocain toxicity[11, 12]. The other important factor is the proper use of ephedrin as a prophylactic against a drop in pressure.

Chen and Smith[13, 14] in their experimental work have come to the conclusion that ephedrin like epinephrin is a sympathomimetic drug, and that its blood pressure raising qualities are produced by its action on the sympathetic nerve endings and by cardiac stimulation through the stellate ganglion. However, De Eds and Butt[15], and Tainter[16] showed that the pressor action is not due to stimulation of any portion of the sympathetic nervous system, but is due to muscular stimulation. Tainter failed to see any stimulation of the stellate ganglion by ephedrin. Therefore, ephedrin like tyramine and phenylaminoethanol belongs to the class of musculo-tropic substances.

Ephedrin in physiological doses is a stimulant. In moderate doses it may become a depressant and at times may cause extrasystoles. Experimental evidence indicates that a toxic dose of ephedrin is 35 to 100 times the physiologic dose. A toxic dose may produce bradycardia, prolongation of the P-R interval, partial A-V block, nodal rhythm, ventricular extrasystoles, bundle branch block, ventricular fibrillation and death[13, 17]. Miller[18] reports the use of

ephedrin in 83 patients with varied pathological conditions. The doses administered varied from 50 to 125 mgs. Of this number he found no change in blood pressure in 8 per cent, a fall in pressure in 7 per cent. The remainder showed a definite rise in blood pressure. In a few cases, 100 to 125 mgs. doses caused extrasystoles, and one case with aortic regurgitation developed a cardiac arrhythmia six hours later. Experimentally it was found that the first intravenous injection of one milligram or more per kilogram of body weight raises and sustains the blood pressure more effectively than later injections which become more ineffective as to degree and duration of pressure elevation. Injection of small doses, repeated before the pressure has fallen from the previous injection will cause a summation in effect[17]. The action of ephedrin on the kidney vessels is first vasoconstriction followed by vasodilatation with a tendency towards increase in urinary output[17, 18].

To prevent vasomotor collapse ephedrin sulphate (Lilly) is given intramuscularly about 15 minutes before the spinal injection. Patients given a low spinal injection not extending higher than the third lumbar segment need very little if any ephedrin. However, it is best for the novice to give a small prophylactic dose of 50 mgs. of ephedrin sulphate as the height of anesthesia is not always so readily controllable and may extend a segment or two higher than was desired. Less ephedrin is needed when the Trendelenburg position is used. In a number of cases for lower abdominal work the Trendelenburg posture with the addition of 50 mgs. of ephedrin have been sufficient protection against a marked drop in blood pressure. If the patient is maintained in the level position during a high spinal anesthesia 75 to 100 mgs. of ephedrin seems to be the best prophylactic dose.

In all instances it is better to have the blood pressure taken before the spinal injection is made. One patient, age 56, in a fair state of nutrition, came under observation with a relatively negative history and physical examination except for inguinal hernia. Eight minutes after the injection of 50 mgs of ephedrin intramuscularly there occurred a 35 mm. Hg fall in pressure and a slowing of the pulse from 72 to 48 per minute. This was an average moderate dose and yet a depressing dose for this particular individual. The operation was performed under local anesthesia. Nine days later 50 mgs. of ephedrin were again given this same patient. He did not react in the same manner, and instead of a drop in pressure there occurred a rise of 35 mm. above the normal reading. The ephedrin action is not always constant, but depends somewhat upon the condition of the patient at the time of the administration of the drug. Each individual has his own susceptability to the drug[19].

The method generally used in this clinic is the administration of 80 to 200 mgs. of novocain crystals (Metz) dissolved in spinal fluid and reinjected. The size of the dose depends upon the weight and the condition of the patient, and the type and length of operation. The height of anesthesia is determined by the selection of the interspace, the amount of spinal fluid used in dissolving the crystals, and the rapidity of injection. In a number of instances when a prolonged anesthesia was desirable, Pitkin's solution "Spinocaine" has been used with equally good results. The aim has been to adhere closely to Pitkin's prescribed technic when using "Spinocaine." Failures in spinal anesthesia are usually due to extradural injection of the anesthetic mixture. A second cause of failure may be an idiosyncracy to novocain. Stout[4] has advanced a technic that controls the height and duration of the anesthesia with the use of novocain and spinal fluid. He is able to produce anesthesia, lasting from 1½ to 2 hours.

After the administration of the anesthetic the patient is placed in the modified Trendelenburg position, whenever it does not interfere with the operation. The danger point lies within the first 15 to 30 minutes following the spinal injection. Patients during this period are very closely watched; the blood pressure, pulse and respiration being taken every

few minutes and recorded. In spite of the prophylactic use of ephedrin there are a certain number of cases which show a drop in pressure. If the pressure remains well within the upper third of the original level there need be little concern. Should the pressure begin to fall rapidly below this level because of previous insufficient ephedrin dosage, the immediate injection of 3 to 5 minims of epinephrin intramuscularly will usually bring up the pressure rapidly. There exists a synergistic action between ephedrin and epinephrin[20].

The early treatment of vascular depression is the best safeguard against vasomotor collapse. The patient during this period responds more readily and effectively to the treatment and the pressure can be kept under control by the subcutaneous or intramuscular injection of a small dose of epinephrin. Once the pressure has fallen to a dangerous level by the above methods, intravenous injections may become necessary. Babcock advises that the fall in pressure to 50 mm. systolic in an asthenic, obese or aged patient, should be immediately counteracted, whereas a drop in systolic to 30 mm. in a young and vigorous patient may require no stimulation. One of the intravenous methods of stimulation recommended by Evans[21] is to give physiologic saline slowly to which has been added 1 minim of epinephrin to 100 c.c. solution. The other method is to give 25 to 50 mgs. of ephedrin intravenously provided excessive doses of ephedrin have not been given intramuscularly. If several doses of ephedrin have been given intramuscularly, it is preferable to administer adrenalin instead of ephedrin intravenously. Wehrbein[22] in emergency, during spinal anesthesia, has used ephedrin intravenously in a few cases with good results. It is almost usless to try to restore the pressure with intramuscular injection of ephedrin as in the presence of a poor circulation its action is slow and very weak. A third method is to give adrenalin intravenously. Pearse[23] has reported two cases in advanced age with hypertension in whom 5 minims of adrenalin were given intravenously at the time of a zero pressure following sacral anesthesia. The recovery was rapid in these aged patients with a temporary rise of pressure above the normal limits followed by a sustaining pressure slightly below their normal level. Nadler[24] has found experimentally that the minimum effective dose of epinephrin is two and one-half times as efficient as ephedrin.

Before the prophylactic use of ephedrin was introduced in this clinic, there was one aged diabetic patient who received the muscular injection of ephedrin while the pressure was dropping. It fell rapidly to a systolic of 40 mm. Hg and remained there. At this stage the patient received 50 mgs. of ephedrin intravenously, and the response was a striking rise in pressure to above normal within 10 minutes. Altogether this patient did not receive more than 100 mgs. of ephedrin.

It is important to use ephedrin cautiously and not to use more than is actually needed, particularly in poor risks and in cases with myocardial disease. Within the last year two aged patients developed a marked irregularity of the pulse with extrasystoles for a period of one hour following the intramuscular injection of 100 mgs. of ephedrin. Sise[1] reports two cases that received 150 mgs. of ephedrin, and in spite of maintaining an elevated pressure developed a small volume pulse, auricular fibrillation and cyanosis, resulting in death in 12 hours. Ephedrin in these poor risks was considered the deciding factor in bringing about the fatal terminations. Large doses of ephedrin when causing a marked rise in pressure for a prolonged period of time may put an overload on the diseased heart. Ockerblad and Dillon[25] have seen no toxic reactions following the use of 300 mgs. of ephedrin.

Before the introduction of ephedrin it was a well known fact that patients eventually recovered from their vasomotor collapse if kept in the Trendelenburg position. Labat has emphasized the importance of this position and still uses it without other stimulation. There are many references in the literature to the value of its use. It prevents the acute

anemia of the brain which results in respiratory disturbances, ranging from air hunger to respiratory failure. The brain is supplied by terminal blood vessels, that are not supported by muscles and aponeurosis as in other parts of the body. Once these vessels are permitted to collapse because of lack of blood, there may occur instant death from respiratory or cardiac failure. Should the blood supply to the brain be impoverished with partial collapse, the patient remains in a comatose or semi-comatose state lasting from 3 to 24 hours. With the use of the Trendelenburg position immediately following the injection such symptoms as pallor of the face with cold sweats and respiratory embarrassment have almost completely disappeared from the picture.

A gas machine should be at hand in case of an anesthetic failure, or in case of emergency for oxygen administration. Evans has found the use of oxygen of benefit in a limited number of cases. In cardiovascular collapse with respiratory embarrassment, evidenced by pallor, cyanosis, loss of voice, and a semi-comatose state, the inhalation of oxygen or oxygen with carbon dioxide serves as a valuable stimulant.

Babcock has outlined a treatment in respiratory depression and respiratory paralysis With colloidan a wisp of cotton is attached to the patient's nose to indicate shallow respiratory movements. If respiration ceases the usual methods of artificial respiration should be instituted. If the compression of the chest fails to move the tidal air in the respiratory passages, then mouth to mouth insufflation with the nostrils and epigastrium compressed becomes necessary. Babcock has saved three patients by mouth to mouth insufflation.

Transient nausea, or nausea and vomiting may occur in a small percentage of patients during spinal anesthesia. Vigorous manipulation of the organs in the abdominal cavity, or traction on the mesentery may excite these symptoms. Patients may become nauseated and vomit after a sudden change in position, or after rough handling in transferring the patient from the table to the cart. Likewise larger doses of ephedrim may cause nausea. In nervous patients, fear may excite these symptoms. If the patient is instructed to breath deeply through the mouth or is given oxygen with carbon dioxide for several breaths these symptoms usually disappear.

When using spinal anesthesia one should have a definite method of procedure and have everything in readiness, so that valuable time may not be lost in controlling the patient's condition. When anesthesia is induced one cannot be certain that there may not be an occasional case of vasomotor collapse. Since the prophylactic use of ephedrin and the institution of early treatment during a falling pressure, no dangerously low pressures have been encountered thus far in this clinic.

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Mother: "Edna, why did you try to deceive Mamma and tell her you didn't eat the cake just because she didn't see you. God saw you."

Edna: "Yes, Mamma, but He didn't talk so much about it."

The Causes of Diarrhea—A Classification for Clinical Use

ALBERT S. WELCH, A.B., M.D.
Department of Medicine

In order to make a correct diagnosis and to treat satisfactorily those conditions in which diarrhea is a prominent symptom, a practical outline of the causes of diarrhea is desirable. The etiologic factors involved are numerous and complex. The outline herewith appended has proven convenient. The main divisions and main subdivisions of the causes of diarrhea are easily kept in mind, and from these groups, diagnoses may be reasoned out in detail, with no necessity of having the entire list memorized.

The commonest causes of diarrhea are: irritating foods or drugs; in conjunction with the upper respiratory infections of children; on an emotional basis; in connection with endocrine and autonomic nervous system imbalance; in old age; in disease entities such as diabetes, nephritis, pernicious anemia, and the deficiency diseases; allergy; with atmospheric changes; and due to infection or infestation of the colon with bacteria or parasites.

At the outset, diarrhea presupposes an intact nervous mechanism of the colon. If the colon were paralyzed, there could be no diarrhea in spite of the accumulation of fluid within its lumen. The nervous impulses responsible for the phenomena of diarrhea may arise either within the colon proper (intrinsic colonic stimulation), or may arise in other parts of the body (extrinsic colonic stimulation).

By diarrhea is meant the frequent passage of stools. An absolute line of demarkation cannot be drawn between normal frequency and undue frequency. There are healthy individuals whose bowels move as often as four times daily. Among the things that must be taken into consideration in determining undue frequency are whether the patient has had frequent passages since birth, if he is inconvenienced physically or mentally by his condition, and whether circumstances exist to which the frequency can be attributed. The character of the stool will throw light upon the situation in a majority of instances.

NORMAL VARIATIONS

The normal stool from a healthy individual varies according to the type of food eaten and the emotional strain to which he is subjected. With the customary articles of diet such as meat, potatoes and green vegetables, an individual whose routine consists of eight hours of ordinary work and plenty of sleep and who is contented will pass a brown formed stool about one inch in diameter. If irritating foods such as cabbage are eaten, the stool normally becomes less formed and is more frequently ejected. If the individual becomes preoccupied, the stool is less frequently passed and is hard and dry. If he is excited, frequent mushy stools may be normally ejected.

It is true that tolerance may be developed, either for extraordinary foods or for routine which would be an emotional strain to the uninitiated. Individuals accustomed to a diet of cabbage may eventually pass a daily formed brown stool without discomfort. If these individuals were then suddenly put upon a bland diet of meat and potatoes, constipation might result because they had become accustomed to the stimulating effect of cabbage. An individual may harden himself to certain routine so that it no longer becomes an emotional strain to him.

A third factor governing the stools in the healthy individual is the climatic environment to which he is subjected. No hard and fast rule can be applied here. Changes in climate may lead to either increased or decreased frequency. The change seems to be the essential factor. Tolerance here may also be acquired.

In regard to the healthy individual and foods, it must not be forgotten that drinks are also part of the diet, water in different communities contains various kinds and proportions of mineral salts which may be either sedative or laxative in action.

Having noted the variations which may occur in healthy individuals due to *food, drink, routine, peace of mind* and *climate,* we may proceed to the pathological conditions with which diarrhea is associated. Ordinarily, readjustment oc-

curs in the healthy individual who meets the variations in life as noted above. But occasionally readjustment is not affected, and this is often the case with individuals whose bodily and nervous constitutions are congenitally unequal to the situations that arise in life; or the situations themselves may be so trying that even the healthy, vigorous individual is eventually overcome.

As an example of the congenitally weak individual may be mentioned one type of school-teacher, thin, irritable, restless, whose exacting daily routine is not met with readjustment, but who seems to suffer with "nerves" more and more as the school season wears on and years succeed one another.

As an illustration of environmental conditions that overcome even the hardy individual may be mentioned the world war in which excellent specimens of men were wrecked by the continued emotional strain to which they were subjected.

Seldom does the diarrhea in itself cause trouble under such circumstances, although it might assume part in a vicious circle, leading to decreased absorption of food and relative starvation, which in turn would lower the physical standard of the individual.

CLASSIFICATION

Intrinsic colonic stimulation may be brought about by (1) mechanical distension of the colon, by (2) chemical irritants, by (3) infection, and by (4) tumors.

MECHANICAL DISTENSION

The colon may become distended with solid, liquid or gas. The distension is a stimulant to the colonic nervous apparatus, and if it is intact, there is a desire to go to stool.

Solid material may be either introduced from without in the form of bulky food such as agar, bran, barium, and green vegetables; or it may grow within the lumen of the intestine in the form of a tumor. Tumors of the colon which by their bulk alone might lead to diarrhea are large intralumenal lipomas, pedunculated carcinomas, and the mass formed by an intussusception.

Liquids as a cause of mechanical distension may be ingested as such or introduced in the form of enemas; they may be generated in the bowel lumen from foods, and they may be excreted from the blood stream into the bowel.

Liquids ingested as such are seldom responsible for diarrhea from mechanical distension. They are usually absorbed in the upper part of the alimentary tract and excreted elsewhere.

Liquids may result from foods that were ingested in a semisolid state, but it is doubtful if diarrhea in such instances can be attributed to the mechanical distension alone. Other factors are usually responsible for the accumulation of liquid in the large bowel such as poor digestion or absorption in the small intestine, too rapid passage of food through the small bowel, or faulty absorption of liquids from the large bowel.

However, liquids excreted from the blood stream into the bowel lumen may through mechanical distension cause diarrhea. Epsom salts influence the passage of fluid from the blood into the bowel lumen; and when fluids are excreted vicariously through the colon in diseases associated with edema, diarrhea may occur.

Gasses even more than liquids, through mechanical stimulation, may lead to diarrhea. Gasses are seldom ingested in sufficient quantity by mouth to reach the colon and bring about mechanical distension great enough to cause diarrhea, even when large quantities are swallowed. Gasses usually develop in the lumen of the alimentary tract from food. While inert gas like nitrogen may be definitely classified as a mechanical stimulant to the colon, the gasses resulting from food decomposition such as acetic acid gas and hydrogen sulphide usually have a chemical irritating effect which is at least in part responsible for the diarrhea that occurs in these particular cases. This will be discussed in detail under the heading, "Chemical Irritants of Intralumenal Origin Produced in the Alimentary Tract from Decomposition of Food."

Gas may also be generated in the lumen of the bowel from diseased bowel wall. Here again other factors, the chemically irritating products of the dis-

ease process and also the inherent lesion of the bowel wall itself, play a part in the diarrhea which results.

Gasses may be excreted from the blood into the lumen of the bowel and cause diarrhea. The mechanical distension is one factor and the chemical effect of the gasses is another. Such conditions may occur in the presence of advanced pulmonary disease with vicarious elimination of gasses via the colon; or after inhaling poisonous gasses, for example, in the deadhouse.

Summarizing the outstanding causes of diarrhea from mechanical distension of the colon, we find bulky inert foods, enemas, large pedunculated intralumenal colonic tumors, intussusception, liquids from the ingestion of Epsom or other salts, and liquids excreted vicariously in conditions associated with edema. It is noteworthy that the cases of diarrhea from mechanical distension of the bowel alone are few in number.

CHEMICAL IRRITANTS

This is the largest group, and embraces the commonest causes of diarrhea. Chemical irritants may reach the colonic wall from within the lumen of the colon, or from the blood stream.

Intralumenal irritants may have been ingested as such, or may have been produced in the alimentary tract either from food decomposition or from the digestive glands.

First let us consider chemical irritants of intralumenal origin ingested as such. These substances may have been taken in the form of drugs whose action is on the bowel throughout its entire length such as mercury, or only locally on the colon after the drug has passed through the small intestine. Many cathartics are in this group such as aloin, rheum, oleum ricini, senna and sodium phosphate. Phenolphthalein presumably acts on the lower part of the large intestine.

The irritants may have been ingested as foods which were inherently irritating or became so through decomposition. Foods which are inherently irritating to the colon are fruits and in particular apples, peaches and plums: complex carbohydrates such as honey, maple syrup or preserves: and certain vegetables, notably rheubarb, cabbage, cucumbers and turnips. Many foods yield irritating substances through decomposition. Some of the toxic material is known as ptomaines.

Second, there are chemical irritants of intralumenal origin produced in the alimentary tract from food decomposition. This decomposition occurs because of (a) faulty digestive juices, (b) hypermotility of the upper alimentary tract, (c) poor absorption of food, and (d) retention of food in the colon.

Any of the digestive juices may be lacking. If there is a large deficit, foods which reach the colon undigested may be sufficiently irritating in themselves to lead to diarrhea.

Diarrhea occurs with achlorhydria. That the achlorhydria alone may be responsible for the diarrhea is doubtful, for many individuals have achlorhydria and no diarrhea. It seems more probable that when diarrhea occurs in the presence of achlorhydria, it is the result of some bodily condition which has caused the achlorhydria, or the result of colonic infestation with parasites which might not have occurred had normal gastric juices been present.

Lack of pancreatic juices undoubtedly leads to lienteric diarrhea. This may be produced experimentally, but clinically so many other things are usually present to complicate the picture that it is exceedingly difficult to prove a given case of diarrhea due directly to the absence of pancreatic juices.

The absence of bile in the intestinal contents is not directly responsible for any diarrhea which may exist.

Hypermotility of the upper alimentary tract results in food reaching the colon before digestion and absorption are completed in the small intestine. Such fecal material may not only be inherently irritating in this condition, but it is also broken down by colonic bacteria and the products of decomposition act as local irritants. The upper alimentary tract may become hypermotile either because of direct stimulation of any part of its wall, or reflexly from conditions present in other parts of the body. Direct stimulation may be from ingested irritants,

from focal ulceration of the mouth, esophagus, stomach or duodenum, or from neoplasm. Conditions which reflexly lead to hypermotility of the entire alimentary tract will be discussed under "Extrinsic Colonic Stimulation."

Poor absorption of food in the upper alimentary tract means that unabsorbed material will reach the large bowel where colonic bacteria will act upon it and the products of decomposition, chemical irritants, may produce diarrhea. There are a number of factors that may be responsible for poor absorption of food in the small intestine. In the first place, the food itself may have properties so that it is with difficulty broken down and absorbed. Such foods are beans, cabbage and bran.

In order that digested foods in the small intestine be absorbed through the lining of the bowel into the blood and lymph stream, the bowel wall itself must be intact. Not only does the health of the bowel wall depend upon absence of local infection of the wall, but also upon an adequate circulation. Thus foods may be poorly absorbed because of an unhealthy condition of the small intestine brought about by diseased bowel wall, circulatory impoverishment in connection with hypertension or passive congestion on a basis of cardiac or pulmonary disease, or lesions associated with the liver which cause portal stasis.

Among diseases of the wall of the small bowel proper may be mentioned typhoid fever, tuberculosis, ulceration, purpura, leukemia, and amyloid disease.

Of the cardiac conditions responsible for poor absorption of food, many could be named including those with valvular incompetence, conduction irregularities, and retrogressive myocardial changes.

On a pulmonary basis, conditions of note responsible for passive congestion of the vicera are extensive fibrosis, solidification of the lobes in pneumonia, and lung compression from intrathoracic air, fluid or tumor.

Portal stasis occurs in advanced cirrhosis, syphilis, tuberculosis and carcinomatosis of the liver and in fatty or amyloid degeneration. Tumors that encroach upon the portal vein may cause stasis.

Food may be poorly absorbed in the lower alimentary tract and decompose to become an irritant because of existing conditions comparable to those outlined in connection with the upper alimentary tract.

Food may be retained unduly long in the colon, with decomposition and the liberation of chemical irritants. This happens when an individual puts off going to stool, or allows himself to become constipated for other reasons. After the first hard fecal mass is expelled, there may be watery material, mucus and tenesmus. Food is retained on account of spasm of the sphincter ani in connection with autonomic nervous imbalance or organic lesions such as anal fissure, fistula, ulcer, tumor or thrombosed hemorrhoidal veins.

Food is also retained with bowel obstruction of the dynamic type: fecal impaction, binding fibrous bands, strictures from syphilis, tuberculosis or carcinoma, from the pressure of a tumor against the bowel—a uterine enlargement, big kidney, inflammatory mass or retroperitoneal sarcoma or hematoma, and from intralumenal polyp.

Bile is a chemical irritant to the large bowel but seldom reaches the colon in an irritating state unless the small intestine is hypermotile.

The second large division under chemical irritants as a cause of intrinsic colonic stimulation is the blood-born toxins. These may have gained access to the circulation through the lungs, skin or alimentary tract. Mercury, arsenic, alcohol and lead are examples, and in botulism toxic material gets into the circulation from the small intestine.

Then, also, toxic material may be generated in the body. With certain deficiency diseases an extensive nonspecific colonic ulceration may occur. Diarrhea is occasionally the most pronounced symptom. Diarrhea may develop in connection with advanced diabetes mellitus, with nephritis, and in pernicious anemia. The toxins are theoretically blood-born, and in these diseases the outstanding symptom, instead of cord changes, edema

or pallor, may be diarrhea.

Diarrhea occurs with extensive burns —perhaps due to nervous reflex, perhaps to toxins reaching the colon via the blood stream.

Summing up the chief causes of diarrhea from chemical irritants, we find cathartics and foods with cathartic properties, decomposed foods; conditions associated with achlorhyhria gastrica; conditions causing hypermotility of the upper alimentary tract, particularly nervous influences; conditions associated with portal stasis such as cardiac decompensation, arteriosclerosis, cirrhosis of the liver, portal thrombosis, dynamic bowel obstruction, the deficiency diseases, diabetes, nephritis, pernicious anemia, and chemical poisons such as mercury and arsenic.

INFECTIONS

These make up an important group causing diarrhea and are more easily understood than most of the others. In many instances the infectious organism may be isolated from the stool and identified, but there are undoubtedly specific infections of the bowel which cause diarrhea, the causative organisms of which have not as yet been positively identified. This latter group must account for some of the cases of disseminated ulcerative colitis that have been termed "nonspecific ulcerative colitis."

Diarrhea may be caused by cocci, bacilli, spirilla, protozoa or worms. While there has appeared but little in the literature in regard to streptococci as the cause of colitis, numerous investigators concur in the opinion that this does occur[1]: and in several cases of so-called "intestinal influenza," both during life and at post-mortem examination, large numbers of streptococci not normally present in the colon have been demonstrated in the stools.[2]

Of the bacilli that cause diarrhea there may be mentioned typhoid and paratyphoid (although constipation may occur just as frequently as diarrhea in these conditions,[3] tuberculosis, *B. fecalis alkaligenes* and *proteus vulgaris.*

The outstanding spirillum responsible for diarrhea is the coma organism of cholera.

Protozoa may cause diarrhea. That *amoeba histolytica* and other forms of amoeba may lead to frequent passages is generally accepted, but there is dispute as to whether or not *trichomonas, chilomastix* and similar organisms may cause diarrhea. The opinion of the author is that these organisms can and do cause diarrhea under certain conditions. Giardia may cause diarrhea.

Among the worms, strongyloides intestinalis[4] may be mentioned in particular as a cause of diarrhea. Tapeworms seldom cause trouble, and round worms ordinarily do not alter defecation unless they cause mechanical obstruction. Pinworms may produce mild intermittent diarrhea.

Summarizing, of the organisms responsible for diarrhea, particular note is taken of amoeba, streptococci, dysentery bacillus, cholera, proteus and strongyloides.

Tumors, through their effect on the bowel wall proper, may cause diarrhea, but in most instances factors other than the mere presence of the mass must be taken into consideration. Such factors are toxic substances produced through the presence of the tumor, damage to the wall of the colon so that there is interference with food absorption, mechanical obstruction to the bowel, and reflex nervous influences from a generally disturbed state of health. Tumors of the colon may lead to diarrhea alternating with constipation, or may produce constipation alone. The tumors may be neoplasm, either benign or malignant, or they may be granulomas.

EXTRINSIC COLONIC STIMULATION

Impulses that lead to diarrhea may arise from stimuli originating in parts of the body other than the colon. For clinical purposes, these may be considered in two main groups: (1) Reflexes from other parts of the alimentary tract and (2) reflexes from parts *other than* the alimentary tract.

From Other Parts of the Alimentary Tract and its Branches. Proceeding from the top of the head downward, the parts of the digestive tube under consideration are the sinuses, middle ear, nasal passages, mouth and its organs, throat,

bronchi and lungs, esophagus, stomach, liver and other accessory digestive glands, diverticuli, appendix and other branches of the intestine. Theoretically, disease of any of these parts could reflexly lead to diarrhea upon certain occasions.

In otitis media, diarrhea may be the most conspicuous symptom, even misleading the pediatrician toward a diagnosis of gastro-enteritis. Of course the general toxic condition plays some part as well as the reflex element, but for practical classification, the diarrhea of otitis media seems to fit in well at this point. The essential thing of course is to recognize the possibility of otitis as a cause of diarrhea, regardless of whether it be reflex or toxic in nature.

In similar manner, pharyngitis and more specific upper respiratory disorders such as measles, diphtheria, or scarlet fever may lead to diarrhea, particularly in children.

There are cases of extensive tuberculous involvement of the lungs with concomitant diarrhea. Other anatomic changes present such as amyloidosis create confusion in attempting to show that the diarrhea is essentially reflex in nature; and for that matter, in most instances several causes may be active.

Diarrhea in connection with pancreatic stones has been described as a particular symptom of that condition. The reflex element must play an important role in this connection.

Bloody diarrhea is a common symptom of intussusception and mesenteric embolism or thrombosis. It may occur also with portal thrombosis.

Parts Other Than the Alimentary Tract. Diarrhea has been produced reflexly through excessive stimulation of the organs of special sense, through autonomic system disorders, cerebral malfunction, spinal disease, and from impulses arising in the pelvic organs or other parts of the body.

(a) Among reflexes arising through the organs of special sense, mention may be made of internal ear stimulation as with sea-sickness; horrifying visual phenomena; disgusting odors; and sickening tastes. Any such excessive stimulation may reflexly lead to diarrhea. Also, excessive stimulation of the skin—a burn, bruising, tickle, may lead to diarrhea.

(b) Most of the above-mentioned reflexes pass by way of the autonomic nervous system. For that matter, the nervous stimuli leading to diarrhea from any cause involve the sympathetic and parasympathetic nerves; but for clinical purposes, a special group is set aside. This group embraces autonomic reflex diarrhea from thermic, atmospheric, allergic, endocrine or chemical influences.

Summer diarrhea is at least partly on a basis of autonomic reactivity to thermic and humidity changes. There are individuals who have severe diarrhea whenever there is a sudden drop in temperature, and visa versa.

Diarrhea has been recognized as an allergic manifestation on many occasions. As an example may be mentioned the case of a girl who had diarrhea every time she ate peanuts. They were "poison" to her.

Loose frequent evacuation is not infrequently a symptom in thyroid disfunction, pregnancy, puberty, the catamenia the menopause and other states in which the endocrine system is unduly active.

Of the chemicals which may give rise to diarrhea, through their action upon the autonomic nervous system, specific mention is made of tobacco, muscarine, adrenalin and pituitrin.

(c) Cerebral disorders which reflexly cause diarrhea may be either so-called "functional," or "anatomical." In various emotional states—fright, sorrow, anger—it may occur. It may be a symptom in mania, dementia, hysteria, or migraine.

The anatomic cerebral lesions that occasionally are accompanied by diarrhea are inflammation, tumor, congenital defect, or pressure.

(d) Similar lesions of the spinal cord or ganglia are sometimes at fault. Diarrhea has accompanied herpes and has been seen with peripheral neuritis.[5]

(e) Stimuli may arise in many parts of the body and cause diarrhea. In the pelvis in particular, such conditions as uterine, ovarian or tubal disorders may reflexly produce this symptom.

Summarizing the chief causes of diarrhea on a basis of extrinsic colonic stimulation or reflex, there are emotional states, encephalitis, meningitis, atmospheric changes, allergic phenomena, endocrine disorders, otitis media and upper respiratory tract disorders, and obstruction of the small intestine.

PRACTICAL APPLICATION

Thus the causes of diarrhea constitute a broad field, embracing definitely known etiologic factors and theoretical possibilities. There are single causes and also diarrheic states due to a number of interacting causes.

However, armed with a knowledge of both confirmed and potential causes, the physician is better equipped to diagnose his case in detail, and of course upon detailed diagnosis competent treatment is based.

There are cases of diarrhea which tax the ingenuity of the medical attendant to the utmost. The routine in such instances is a detailed history and complete physical examination, any laboratory work tending to throw light on the patient's general condition, and then retrospective study of the fine points in the patient's history. Meanwhile, the routine treatment for diarrhea in general may be started; but the physician should never lose sight of the fact that this treatment is empirical and never curative except by accident. Chronic diarrhea is a serious symptom and demands much study. To the well-trained physician, there is no end of research possible in each case, and the point is never reached at which he may truthfully say, "I have exhausted all means of study and am at the end of my rope." Accurate diagnoses and cures have been established even after years of persistent observation and experimentation.

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TUBERCULOSIS ABSTRACTS

Proposals for the treatment of tuberculosis by some form of nutritional therapy reappear periodicaly. Recently, the regimen devised by Gerson, based on the studies from Sauerbruch's clinic in Munich, has attracted the attention of physicians and laymen, largely through incomplete press reports of a "salt-free diet." Edgar Mayer of Saranac Lake was privileged to visit Sauerbruch and Gerson's clinic to observe their dietary methods. Upon returning to this country, Dr. Mayer and his associates, with the assistance of Frau Jungklaus who received her dietetic training in the German clinic, applied the method to a group of 30 patients. The results are reported in the Journal of the American Medical Association, December 14, 1929, from which the following abstracts are derived.

BASIC (VITAMIN) FEEDING IN TUBERCULOSIS

The diet in advanced tuberculosis should be of a caloric value sufficient to cover the metabolism (increased by the toxemia especially) as well as the atrophy induced by the disease. But the pulmonary mechanism should not be burdened by an excessive caloric intake. Fifty calories per kilogram is the optimum. Theoretically, the protein intake should be increased, but this accelerates metabolism and hence prevents pulmonary rest. On a 3,500 calory diet, a daily intake of 100 grams of protein will maintain a tuberculous patient in positive nitrogenous equilibrium.

GERSON DIET IS BASE-FORMING

Fulfilling the caloric requirements is not enough for the wasting in tuberculosis involves a corresponding demineralization. The Gerson diet makes good this deficiency by including base-forming solids. The German physicians seem to be confused about the acid-forming and base-forming values of the dietary, but Mayer states that "an analysis of the diet reveals definitely its base-forming nature." Sodium chloride is excluded because it favors tissue hydration, which is already excessive in tuberculous processes. The most significant feature of the dietary is that it is rich in vitamins A to G, and this constitutes a spe-

cific advance in this particular form of dietary treatment of tuberculosis. A sample diet for one day is shown in the table.

Mayer observed in the German clinics "definite healing in advanced cases of lupus vulgaris of the face and mucous membranes that had previously been resistant to all other accepted forms of treatment. These results seemed far more striking to us than those claimed for the other forms of tuberculosis, which included tuberculosis of the bones and joints, lymph nodes, lungs, peritoneum and genito-urinary tract, both with and without sinuses." Lupus is a prevalent complication in European countries, while in the United States it is relatively rare.

DIET TESTED IN UNITED STATES

Upon returning to this country, Mayer selected 30 patients who had failed to respond to accepted theraueptic measures for two or three years. Of these, 10 were excluded before the end of the six months' period for various reasons. The 20 patients who completed the experiment showed a substantial gain in weight which it had been impossible to attain by previous procedures. Many of the alimentary disturbances, which had persisted on other diets, cleared rapidly with this regimen. The diet was well tolerated except in two instances. It was effective in diminishing fatigue and induced a sense of well-being. The slight elevation of temperature disappeared in a few cases.

Physical and roentgenological examinations of the chest showed in about one-third of the cases definite clearing, with an occasional contraction of a cavity. The other patients did not show any marked changes. The quantity of sputum diminished considerably in about half the cases, but in none did it become negative for tubercle bacilli. A few of the patients showed marked diminution of cough. Hemorrhages occurred in some during the experiment as well as before. Chest pains disappeared in a few who constantly complained of this symptom before. Two cases of intestinal tuberculosis which had resisted other therapeu-

Rats fed on acid-forming (left) and base-forming (right) diets.

tic procedures cleared up clinically, and the roentgenological filling defects of the colon disappeared; one remained unchanged.

EXPERIMENTS ON RATS

The author cites also chemical and nu-

Sample Diet for Tuberculosis Patient for One Day

3,500 calories; 70 Kg.; 1 Gm. per Kg. of body weight; basic, 40 cc.

7 a. m.		
Oatmeal	4 T	1⅔ oz.
Sugar	1 T	⅓ "
Cream	2 T	1 "
8 a. m.		
Br. tomatoes	1	3½ "
Stale bread	1 sl	1 "
Sweet butter	2 pats	⅔ "
Cream cheese	1" cube	½ "
Honey	1 T	½ "
Milk	1 C	8 "
11 a. m.		
Lemon juice	½ C	4 "
Sugar	2 T	1 "
Egg yolk	1	⅔ "
Butter	1 pat	⅓ "
Crackers	2	½ "
1 p. m.		
Veg. soup	1 C	8 oz.
Potato	1	5 "
Peas	Serv	3½ "
Carrots	Serv	3½ "
Lettuce	Serv	1⅔ "
Mayonnaise	1 T	½ "
Bread	1 sl	1 "
Butter	2 pats	⅔ "
Milk	1 C	8 "
4 p. m.		
Cocoa	1 C	8 "
Bread	1 sl	1 "
Butter	1 pat	⅓ "
6 p. m.		
Rice	2 T	1 oz.
Lettuce	Serv	1⅔ "
Cheese	1" cube	½ "
Bread	1 sl	1 "
Butter	1 pat	⅓ "
Milk	1 C	8 "
8 p. m.		
Oatmeal	4 T	1⅔ "
Sugar	1 T	½ "
Cream	2 T	1⅓ "
Almonds		⅓ "

In addition, the patient receives one tablespoon cod liver oil three times daily, much fruit between meals and about five grams of meat weekly.

tritional studies in two groups of albino rates to determine the effect of acid- and base-forming diets on the blood, bones and metabolism. Compared with the rats on the acid-forming diet, those on base-forming diets gained markedly in weight, their bones were larger, firmer and non-rachitic, their muscular tone was better, they were more vigorous, their appetite was keener, they did not succumb to infection as easily; in short, the general metabolism and well-being was superior.

Dr. Mayer realizes that this preliminary study of human patients permits of no dogmatic statement because the group was distinct from those of the German clinics, where more favorable results were observed, and because of the difficulty of clinic control; the only means being the selection of patients who failed to respond to accepted therapeutic measures for two or three years. The favorable results in about one-third of the patients studied for a period of six months of careful supervision may perhaps be attributed to the effect of the dietary. But critical clinicians may justly maintain that contributory factors other than diet were operative in the end-result. The psychic element, the enforced rest, the occasional tendency of the disease to subside spontaneously, the whole-some food ,its scrupulous preparation and careful cooking, and the individual service are factors that must be considered in a final analysis.

IMPORTANCE OF VITAMINS

Mayer believes that the inorganic constituents, whether acid-forming or base-forming, are not as important as the vitamin content of the dietary. Such foods as milk, vegetables, and fruits are rich in vitamins and are base-forming. These two factors regulate cellular metabolism, and the utilization of minerals depends on the simultaneous presence of vitamins in the alimentary tract.

Extravagant and unwarranted claims have been made for the Gerson diet but it is certainly not a cure for tuberculosis. In isolated cases, it may be of value but only as an aid to the accepted routine treatment whose mainstay is still rest.

MEDICAL SCHOOL NOTES

Dr. J. E. Ackert, chairman of the graduate council at K. S. A. C., addressed the medical students of the University of Kansas, Wednesday, February 26, on "Important Human Parasites."

"With much new equipment and a staff of eminent specialists, Kansas has reasons to be proud of her University Medical School," Dr. Ackert said, when speaking of the new building in which the Medical School is housed.

Dr. Earl C. Padgett and Dr. C. B. Francisco recently spoke to the County Medical Society of Chanute, Kansas Their subjects were "Sacro-iliac Sprain" and "Some Phases of Plastic Surgery."

The first course of lectures under the Porter Lectureship in Medicine was given by Dr. Lewellys F. Barker, Professor Emeritus of Medicine of the Johns Hopkins Medical School on April 1 and 2. In 1918, Dr. J. L. Porter of Paola, Kansas, bequeathed to the Medical School a sum of money for the stimulation of scholarship and research in the Medical School. A portion of the income from this fund has been used to provide a scholarship for a worthy student. The remainder of the income is to be used to defray the expenses of an annual lecturer in medicine.

Dr. James H. Danglade, '26, an instructor in the department of medicine of the Johns Hopkins Medical School, visited here recently.

Dean H. R. Wahl and Dr. Ralph H. Major attended the annual meeting of the Council on Education of the American Medical Association held in Chicago recently.

Intensive graduate courses in obstetrics, gynecology, pediatrics and ophthalmology have been given for practitioners over the state. Outstanding specialists from various medical schools over the country have served on the faculties for the courses.

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THE MEETING

The most successful annual meeting in the history of the Society has just closed. According to the registration books there were 512 in attendance. The convention hall was crowded to its capacity on Wednesday which was "Guest Day." There were no vacancies on the program, of any day, every man was on hand with his paper or lecture at the proper time. A full report will be printed in the June number.

THE BRINKLEY MATTER

Now that the complaint against Brinkley has been filed and he has been regularly summoned to appear before the State Board of Registration and Examination to show cause why his license to practice should not be revoked the editor is at liberty to spill all the beans he wishes. However, much of what could be said is now past history and the newspapers have already made it public.

It is the most natural thing in the world that the members of the Society should become impatient with what seemed to them unnecessary indolence in the disposition of such an intolerable situation.

The wisest council was against publicity until such time as the preparations for procedure were completed. It seemed to many that there was sufficient evidence of fraud and of gross immorality to revoke the license of any practitioner, of any other practitioner at least. But this man not only had friends in high places but he had resources for publicity by which he was able to form public opinion to his needs. A man of less notoriety, less keenly alive to the precarious position he held, and less strongly defended by the religious, political and commercial influences his foresight had created, would have been hailed before the board and deprived of his license in short order. But in this case it was very essential to be sure that the action of the board should stand the test of the highest courts for it was conceded that the action of the board would be so contested. Since in such a case the board must be, or is supposed to be, defended by the Attorney General it was regarded as of considerable importance that before any action was begun he should be satisfied of the extent, nature and sufficiency of the evidence. Perhaps it was one's impatience that led one to believe the Attorney General was too hard to satisfy.

Comparing the evidence already accumulated with the evidence upon which other licenses have been revoked there is hardly room for the slightest doubt how the board will act in the matter. After that we will perhaps have an opportunity to see what attitude the Attorney General will take. A great deal of credit should be given the Kansas City Star for its exposure of the practices at the Brinkley hospital and it is freely conceded that the Star was largely responsible for the final yielding of the Attorney General to the demands for immediate action.

COST OF ILLNESS

During the past year there has been an unusual amount of discussion in both

lay and medical publications on the high cost of medical care in which both the medical profession and the hospitals have been subjected to considerable criticism. There has often been a considerable element of hysteria and misinformation on both sides of the argument. Generalizations are frequently made on the basis of isolated and unusual instances that on careful analysis are manifestly unfair and exceptional. The patient will complain of the exorbitant charges that a certain doctor has made "without doing anything" and the physician will tell of a shabbily dressed man coming to the dispensary in a Packard sedan for free treatment. The patient states that the hospital charges are robbery and the nurses have no interest in their welfare and the hospitals feel that the patient should have selected cheaper accommodations and excuses its high charges on the basis that the doctor on the case has ordered so many special tests and expensive therapy. The patient feels that his doctor is no longer particularly interested in his welfare and the doctor holds that since the patient has spent too much money on patent medicines and irregular pracitioners that, after his years of intense study and starvation, he is entitled to a reasonable fee for his time and advice.

The medical profession is not so well thought of by the laity now as it was a generation ago when the average physician was a "family doctor." It is quite probable that there is some exaggeration of this supposed lack of confidence yet it must be admitted that there is some truth in it. This is the natural result of the increasing specializaion of medical practice. There is no longer such an intimate contact between the patient and the doctor in many instances and both parties are apt to acquire a warped impression of each other.

The average man who goes to a free clinic goes by necessity and not by choice. This is evident to anyone who has been in a large free clinic and who has taken time to inquire into the circumstances of these clinic patients. Less than 5 per cent take advantage of such free treatment. Many tragic situations are revealed in such inquiries. The average man resents the imputation of accepting charity and wants to pay if he can. Often he is misinformed or deluded as to the nature of a free clinic and doesn't know that he is making unfair demands on a doctor's time.

Hospitals are often accused not only of being expensive but being cold blooded and enmeshed in red tape. The average patient entering a hospital has the no tion that he is the only one in the institution and that everything must be done to please him. He doesn't realize that what he wants may be contrary to his doctor's orders or may be detrimental to other patients who may be near him. The "so-called red tape" is provided to secure obedience to the physician's orders and to provide the welfare of the great majority of patients in the institution. This may interfere with the desires of an individual patient and, often to save the doctor, the hospital takes the blame.

The fact is that the cost of medical care is not high when considered in the light of its importance, the trend of modern times nad the price the public pays for its luxuries. It has been estimated that the total cost of medical care in the United States during the past year was a little less than three billion out of a total income of 90 billion dollars. Health is of the greatest importance to the public yet the cost of its maintenance is much less than what it expends for candy, tobacco, the motor car or the movie. There is no public clamor against the high cost of candy or tobacco!

Co-operation between the doctor and the patient can do much to lower the cost of illness. Physicians often order an unnecessary number of special tests and examinations that could be eliminated if they would rely more on their five senses in making the diagnosis. Many patients could reduce their expenses if they would not go to a specialist for minor ailments but rely more on the general practitioner, if they would stop wasting money on patent medicines, quacks and cultists and if, when they enter a hospital, be willing to accept the less expensive beds. Many patients fail to recognize that the medical treatment in the wards is fully as good and much less expensive than that in private rooms.

Much of the recent discussion of health costs is permeated with misinformation. The medical profession should see to it in the future that the public is not only kept informed of the advances of medical science but is persuaded to accept the application of these advances. Physicians generally give free information to each other of their results and discoveries but make little active effort to make the public understand their value. The medical profession has been remiss in acquainting the public with what it can do with disease. Not only should the profession educate the public but should set as its goal in the next decade, the availability of everything medical science has to offer to every person regardless of his social or economic status and education. At present it is generally recognized that this is accessible to the rich and the poor but the great self respecting middle class deserves such education and such service without being forced to the alternative of accepting charity or receiving inadequate service.

Much of the dread of illness arises from the fact that it catches the average man unprepared and the economic crisis resulting therefrom is apt to give him a warped perspective of the medical profession and of hospitals. Hence, second to the education of the public with what medical science can do for it, the medical profession should encourage some form of health insurance so that when the inevitable illness develops, it does not become such a financial burden.

In the third place the profession should urge the public to consult their family physician more frequently and make fewer calls on the more expensive specialist, inasmuch as a good general practitioner is fully competent to take care of 85-90 per cent of all types of illnesses requiring medical advice. If this is done, there is no danger of the "family doctor" disappearing. The only obstacle in the desired maintenance and rehabilitation of the family physician is the feeling that the family doctor may sell his patient to the highest bidder rather than to send him to the most competent man that is available. This is the danger of the "fee splitting evil" and the reason why the more thoughtful members of the profession are making such efforts to clean this evil out of the profession.

H. R. Wahl.

SOCIETIES

RUSH-NESS COUNTY SOCIETY

The Rush-Ness County Society met in LaCrosse, Kansas, April 7th with Dr. F. D. Smith. There were nine members present. Dr. W. Singleton of McCracken presided. Reports of secretary and treasurer were read and approved.

On a motion regularly seconded and carried the Rush-Ness County Medical Society went on record as offering a protest against the activities of one J. R. Brinkley of Milford, Kansas, and his radio broadcasting over station KFKB in that his conduct was unethical and unprofessional and that his statements

were false, vulgar and misleading to the public.

Dr. H. C. Embry of Great Bend, Kansas, gave a very interesting, as well as instructive, paper on Intestinal Stasis and its relation to arthritis. The paper reported the results of some of our leading authorities as well as some of Dr. Embry's own cases; giving us a newer etiological factor to consider in this type of cases.

Dr. L. A. Latimer of Alexander was elected state delegate and Dr. W. S. Grisell as alternate, to the state meeting in Topeka, Kansas, in May.

Dr. H. R. Bryan of Hayes, Kansas, was a guest of our Society and he will furnish us with a paper at our next regular meeting with Dr. Grisell at Ransom, Kansas, in June.

F. D. Smith, M.D., Sec'y.

ROUBBON COUNTY MEDICAL SOCIETY

Bourbon County Medical Society met in the Library Building in Ft. Scott, Kansas, at 8:00 p. m. April 21, 1930, with Dr. W. S. Gooch, the president, in charge. There were fourteen doctors present.

A motion was made and seconded, that a letter be sent to the Hon. Gov. Reed, favoring the re-appointment of Dr. A. S. Ross and Dr. A. D. Gray, as members of the State Board of Medical Examiners.

A committee, representing the society, was appointed to draw up resolutions, recommending the State Board of Medical Examiners, to revoke Dr. J. R. Brinkley's medical license to practice medicine because of unethical practices.

Dr. Ray M. Balyeat, of Oklahoma City was the guest speaker and gave a very excellent paper on allergy, including asthma, hay fever, urticaria, eczema, and migraine. He illustrated with lantern slides. His lecture was greatly appreciated. Meeting adjourned at a late hour.

The Bourbon County Medical Society wishes to submit the following resolutions to the Honorable Board of Medical Registration:

Whereas, The Bourbon County Medical Society wishes to express their disapproval of the professional conduct of one John R. Brinkley of Milford, Kansas, and

Whereas, Said John R. Brinkley was at one time made a member of our county society we wish to express our regrets that he was ever recognized by us as a fellow, and

Whereas, The Bourbon County Medical Society wishes to recommend to our Honorable Board of Medical Registration that the license of said John R. Brinkley be revoked, and be it further

Resolved, That a copy of these resolutions be sent to the Journal of the Kansas State Medical Society.

On motion of Dr. J. D. Hunter, seconded by Dr. R. O. Crume after discussion, was unanimously adopted.

Committee: Ralph O. Crume, M.D.; John D. Hunter, M.D.; W. T. Wilkening, M.D.

R. Y. Strohm, M.D., Sec'y.

DICKINSON COUNTY

The Dickinson County Medical Society met in Herington, Kansas, as guests of the Herington doctors. Twenty-one doctors were present including a few from Morris and Marion counties. Supper was served at the Hotel Daily. A motion picture was shown on "The Relation of Absorbable Suture to Wound Healing" furnished by Davis and Geck, Inc. Dr. Victor F. Chesky of Halstead read a paper on "Gall Bladder Disease." Both paper and reel were much enjoyed.

A resolution was passed expressing commendation to the Kansas City Star for its praiseworthy efforts in exposing the lies, misrepresentations and unethical methods used by Dr. J. R. Brinkley of Milford, Kansas, in roping in innocent victims for the purpose of defrauding them of their money. Requests were made to the Attorney General of Kansas and the Federal Radio Commission, that on the basis of the evidence published in the Kansas City Star, steps be taken to have Brinkley's license to practice revoked and that he be prohibited from the further use of his broadcasting station in the promotion of his medical bunk.

The next meeting will be at Hope, Kansas.

DANIEL PETERSON, M.D., Sec'y.

CLAY COUNTY MEDICAL SOCIETY

The April meeting of the Clay County Medical Society was held in Clifton, Kansas, on the evening of the 16th. The members of the society were guests to a dinner at the McCoy hotel which was provided by Drs. Scott, Van Scoyoc and Potter of Clifton.

Following the dinner the society met in Dr. Van Scoyoc's office for the business and scientific sessions.

A number of communications were read and the regular business of the society taken care of.

Applications for membership into the society from Dr. T. C. Kimble, Miltonvale, Dr. L. J. L'Ecuyer, Greenleaf, Dr. Z. H. Snyder, Greenleaf, and Dr. L. J. Weiss, Clay Center, were voted upon and all were elected to membership in the society.

The proposition of a full time health officer for Clay County was brought up and it was moved, seconded and carried that the Society go on record as favoring a full time man and further that a committee of three be appointed to interest the civic clubs of Clay Center and other organizations of the county in the movement. Dr. H. E. Potter, acting president, appointed on this committee Drs. R. W. Diver, E. N. Morgan and E. N. Martin.

Following the business session, Dr. S. T. Millard gave a talk on "Pre-cancerous Skin Lesions" which was very instructive and enjoyed by all present. He also presented some dermatological cases and discussed them.

Due to the inclement weather, the attendance was not up to normal as only thirteen members were present.

The society will be guests of Drs. Carr and Smiley at Junction City for the May meeting.

F. R. CROSON, Sec'y.

JEWELL COUNTY SOCIETY

Jewell County Medical Society members and their wives or husbands were guests of Dr. and Mrs. Hawley at the M. E. Church of Burr Oak on April 22. Luncheon was served in the dining room of the church during which time music was furnished by the Burr Oak orchestra.

At one o'clock Dr. C. S. Kenney of Norton gave an address which was open to the public. The Burr Oak schools attended in a body. Immediately after his address Dr. Kenney held a T. B. clinic at Dr. Hawley's office. A brief business meeting was held by the members, after which Dr. Yankey of Mankato read a paper on "Undulant Fever." A discussion followed.

The members showed their appreciation of Dr. and Mrs. Hawley's hospitality by giving them a vote of thanks for the very pleasant time spent together.

C. W. INGE, M.D., Secretary.

FRANKLIN COUNTY SOCIETY

The Franklin County Medical Society met in regular session at Garnett, Kansas. This was a joint meeting with Allen County and Anderson County Medical Societies. Dinner was served by the ladies of the United Presbyterian Church. There were thirty-four participants.

Dr. Wm. T. Tilley who lately located with us was taken into membership. A resolution to commend the work of A. B. McDonald and the Kansas City Star regarding John R. Brinkley was made a matter of consideration and received the unianimous vote of the three joint societies. Accordingly the following letter was drafted and ordered sent to Mr. McDonald:

"The Franklin, Allen and Anderson County Medical Societies in joint session, April 30, by a unianimous vote commend your action in the John R. Brinkley broadcasting quack affair.

"These three societies congratulate you on the results so far obtained and have confidence that you will continue to use your almost unlimited opportunity to suppress quackeries, charlatanisms and cupidities that prey on the credulity of invalidism."

Talent for the program was all present. Dr. Terrill gave a very practical paper—a resume of his last half year's experience with scarlet fever. There was an extensive and very practical discussion

of the disease from its various angles. Dr. Terrill himself led all those that were already committed to the serological treatment.

The second half of the program took for its theme a paper by Dr. P. S. Mitchell of the Allen County Society. The subject of the paper was "What is Mine," an original thesis that required much study, learning and thinking by the author. He led us far afield. Away from the beaten paths of medicine and surgery. Through unfrequented lanes, beside the quieter shrines of metaphysics and psychology toward the realms of the spiritual. Withal, pointing out along the way many things, by us hitherto unseen or unnoticed. Iconoclastic. The good doctor tried to eliminate "mind" as an entity; indicating as best this writer could understand that what we so easily called mind was not a separate entity but, rather, a functioning normal or abnormal, of brain, nervous system, musculature and fluids of the body.

Discussion of the paper was opened by Dr. T. B. Homan, Prof. of Psychology, Ottawa University. His discussion and the paper led us all out into speculative questionings and opinionations commensurate with our several experiences up to our several mile posts along the journey of life. Dr. Mitchell's paper was by common concensus of opinion classed among the best things ever offered to our societies.

A resolution was unanimously passed by the joint societies requesting the writer to submit the paper to the Kansas State Medical Journal for publication.

GEO. W. DAVIS, M. D., Sec'y.

Clinical Conference of the St. Louis Clinics

The St. Louis Clinics will depart from its usual procedure in conducting post-graduate courses in the fields of medicine and surgery. The Clinical Conference which will take place in St. Louis, Missouri, June 9 to 21, inclusive, will consist of a series of lectures, demonstrations, clinics and round-table luncheon discussions on medical and surgical subjects of interest to the general practitioner. No attempt will be made to arrange the material in special courses, but it has been so selected and arranged that practically all fields of medicine, surgery and allied subjects will be included.

St. Louis is fortunate in the possession of two outstanding medical schools of the country with a wealth of unsurpassed clinical material. The present conference is offered to give the profession an opportunity of coming into close contact with this valuable material and the many excellent local clinicians.

The subjects have been selected with the specific idea of assisting the practitioner who wishes to refresh his mind on many of the common things which are met with in general practice. The luncheon round-table discussions which will be participated in by several clinicians noted in their particular fields should be very interesting and most valuable. In these discussions an attempt will be made to carry out the symposium idea so that the subject will be completely covered.

Clinicians of national and international prominence have accepted the invitation to participate in the conferences. This type of clinical conference has a distinct place in post-graduate medical teaching. We congratulate the St. Louis Clinics upon the recognition of this idea and upon inaugurating the conference which doubtless will be an annual event.

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BOOKS

The Medical Clinics of North America. (Issued serially, one number every other month) Volume 13, Number 5. (Chicago Number, March 1930.) Octavo of 207 pages with 17 illustrations. Per clinic year, July 1929 to May 1930. Paper, $12.00; Cloth, $16.00 Net. Philadelphia and London; W. B. Saunders Company, 1930.

Williamson and Birch present an analysis of the wrong diagnoses in four years of medical service as revealed by autopsy. This is particularly interesting because of 128 cases so studied 119 were diagnosed correctly. Among the instructive papers in this volume are: Disorders of sleep by Pollock; periarteritis by Carr; the vomiting mechanism in the early toxemias of pregnancy; undulant fever by Strause and Howell; appendicitis in children by Gertsley; heart failure by Brams; uremia in children by Calvin; the use of digitalis in the control of auricular fibrillation, by Fenn; the relation of headache to upper abdominal distress. There are other clinics in which every practitioner will be interested.

Surgical Diagnosis. By 42 American authors. Edited by Evarts A. Graham, M.D., Professor of Surgery, Washington University Medical School. Three Octavo volumes, totalling 2,750 pages, containing 1,250 illustrations, and separate index volume. Philadelphia and London: W. B. Saunders Company, 1930. Cloth, $35.00 a set. Volume I and II are now ready. Volume III and separate index volume ready March 15, 1930.

There is need for a comprehensive work on surgical diagnosis, even if nothing particularly new is brought out. Simply to make conveniently available all the knowledge on the subject is eminently worth while. With such a large list of the outstanding surgeons of the country contributing to this work it should meet the demands for the present at least. The editor of the work suggests that patients are frequently operated upon with too little preliminary study—that is before a correct diagnosis has been made. If better facilities for making correct diagnoses will tend to mitigate such an unfortunate condition of affairs this publication will certainly accomplish much. The work is well illustrated.

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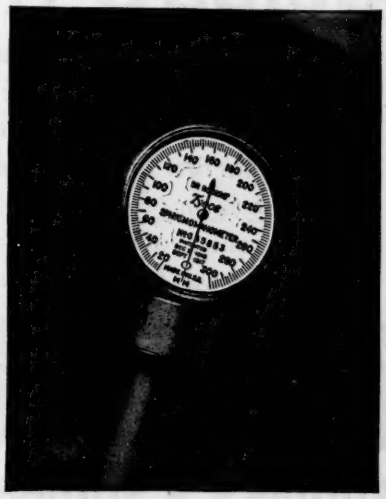

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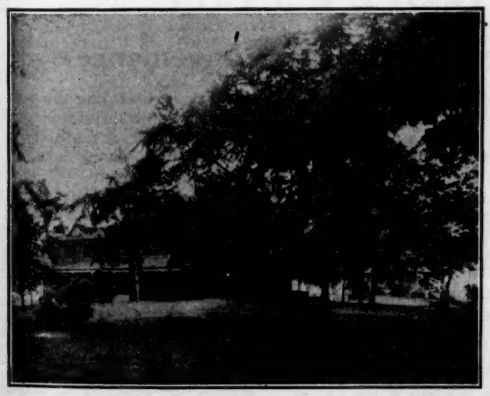

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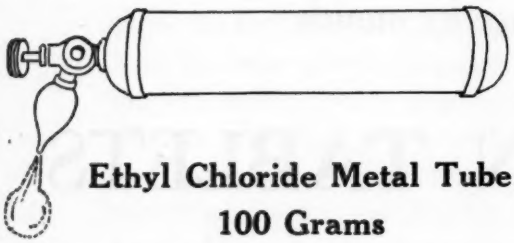

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Committee on Necrology—E. E. Liggett, M.D., Chairman, Oswego; J. F. Hassig, M.D., Kansas City; W. E. McVey, M.D., Topeka.

Members of the Component County Societies are members of the Kansas Medical Society. Physicians residing in counties where no County Society exists may join the society of an adjoining county. Physicians residing where no County Society exists, who are members of a district or other independent society approved by the Council, may be admitted to membership.

ANNUAL DUES $7.00, due on or before February 1st of each year.

Dues should be paid to the Secretary of the Component County Society, or, if not a member of a County Society, to the Secretary of the Kansas Medical Society.

OFFICERS FOR 1930

COUNTY	PRESIDENT	SECRETARY	MEETINGS HELD
Allen	R. O. Christian, Iola	P. S. Mitchell, Iola	
Anderson	W. K. Johnson, Garnett	J. A. Milligan, Garnett	2nd Wednesday
Atchison	M. T. Dingess, Atchison	T. E. Horner, Atchison	1st Wed. ex. July and August
Barton	T. J. Brown, Hoisington	L. R. McGill, Hoisington	1st Tuesday, Jan., Apr., June, Oct.
Bourbon	W. S. Gooch, Ft. Scott	R. Y. Strohm, Ft. Scott	2nd Monday
Brown	H. J. Deaver, Sabetha	S. M. Hibbard, Sabetha	2nd Friday
Butler	G. G. Whitley, Douglas	J. M. Devereaux, El Dorado	2nd Friday
Central Kansas	L. V. Turgeon, Wilson	F. K. Meade, Hays	Dec., March, June, Sept.
Cherokee	R. C. Lowdermilk, Galena	W. H. Iliff, Baxter Springs	2nd Monday
Clay	C. C. Stillman, Morganville	F. R. Croson, Clay Center	2nd Wednesday
Cloud	Ellis Starr, Concordia	R. E. Weaver, Concordia	Last Thursday
Coffey	H. T. Salisbury, Burlington	A. B. McConnell Burlington	
Cowley	C. O. Hawke, Winfield	F. K. Torrence, Winfield	1st Tues. ex. July, Aug., Sept.
Crawford	R. M. Markham, Pittsburg	C. H. Bendage, Pittsburg	3rd Thursday
Decatur-Norton	C. S. Kenney, Norton	W. Stephenson, Norton	Called
Dickinson	H. Marshall, Herington	D. Peterson, Herington	
Doniphan	A. E. Cordonier, Troy	W. M. Boone, Highland	1st Tues. Jan., Apr., July, Oct.
Douglas	A. J. Anderson, Lawrence	L. S. Powell, Lawrence	1st Thursday
Elk	R. C. Hutcheson, Elk Falls	F. L. DePew, Howard	Called
Finney	C. Rewerts, Garden City	O. W. Miner, Garden City	
Ford	C. E. Bandy, Bucklin	W. F. Pine, Dodge City	Last Wednesday
Franklin	Wm. J. Scott, Ottawa	G. W. Davis, Ottawa	
Harper	C. E. Ressler, Anthony	A. E. Walker, Anthony	3rd Wed., Mar., June, Sept., Dec.
Harvey	Arnold Isaac, Goesset	M. C. Martin, Newton	1st Monday
Jackson	E. W. Reed, Holton	C. A. Wyatt, Holton	1st Wed., Jan., Apr., July, Oct.
Jewell	J. E. Hawley, Burr Oak	C. W. Inge, Formosa	
Johnson	C. W. Jones, Olathe	D. E. Bronson, Olathe	Second Monday
Kingman	C. W. Longenecker, Kingman	H. E. Haskins, Kingman	
Labette	O. E. Stevenson, Oswego	J. T. Naramore, Parsons	2nd Thursday ex. summer months
Leavenworth	Leon Matassarin, Leavenworth	H. J. Stacy, Leavenworth	1st Monday
Lincoln	H. L. Hinkley, Barnard	M. Newlon, Lincoln	1st Monday
Linn	D.E. Green, Pleasanton	H. L. Clark, LaCygne	2nd Thursday
Lyon	Albert Beam, Americus	P. W. Morgan, Emporia	2nd Wednesday
McPherson	W. C. Heaston, McPherson	G. R. Dean, McPherson	1st Tuesday
Marion	E. H. Saylor, Marion	E. H. Johnson, Peabody	2nd Wednesday
Marshall	R. L. McAlister, Marysville	H. Haerle, Marysville	Last Thurs.. July, Oct., Jan., Apr.
Meade-Seward	G. S. Smith, Liberal	E. Trekell, Liberal	Second Tuesday
Miami	O. C. Lowe, Paola	J. F. Fowler, Osawatomie	
Mitchell	W. H. Cook, Beloit	Martha Madtson, Beloit	
Montgomery	J. B. Blades, Independence	J. A. Pinkston, Independence	1st Tuesday
Nemaha	F. S. Deem, Oneida	S. Murdock, Jr., Sabetha	
Neosho	J. A. Butin, Chanute	A. M. Garton, Chanute	Last Thursday every other month
Osborne	J. D. Johnson, Alton	S. J. Schwaup, Osborne	Second Monday
Ottawa	L. M. Hinshaw, Bennington	C. M. Vermillion, Minneapolis	
Pawnee	F. W. Ewing, Larned	Glen Weaver, Larned	
Pratt	E. M. Ireland, Coats	W. F. Bernstorf, Pratt	2nd Tuesday
Reno	F. W. Koons, Nickerson	C. A. Boyd, Hutchinson	1st Monday
Republic	M. D. McComas, Courtland	H. E. Robbins, Belleville	4th Friday
Rice	C. C. Price, Lyons	C. W. Haines, Little River	2nd Thursday in November
Riley	G. A. Cassidy, Manhattan	H. T. Groody, Manhattan	First Monday
Rush-Ness	W. Singleton, McCracken	F. D. Smith, LaCrosse	First Monday
Saline	J. E. Miler, Salina	W. R. Dillingham, Salina	
Sedgwick	C. H. Briggs, Wichita	F. H. Schiltz, Wichita	1st and 3rd Tuesdays
Shawnee	A. D. Gray, Topeka	E. G. Brown, Topeka	1st and 3rd Tuesdays
Smith	D. W. Relihan, Smith Center	V. E. Watts, Smith Center	First Monday
Stafford	M. M. Hart, Macksville	R. E. Stivison, St. John	Second Thursday
Sumner	W. A. Heap, Mulvane	I. H. Dillon, Wellington	2nd Wednesday
Washington	H. D. Smith,Washington	W. M. Earnest, Washington	Last Thursday every quarter
Wilson	C.H. Dewey, Elk City	E. C. Duncan, Fredonia	
Woodson	S. E. Bamford, Yates Center	H. A. West, Yates Center	Second Monday
Wyandotte	L. L. Bresette, Kansas City	L. G. Allen, Kansas City	Every 2nd Tues. ex. summer months

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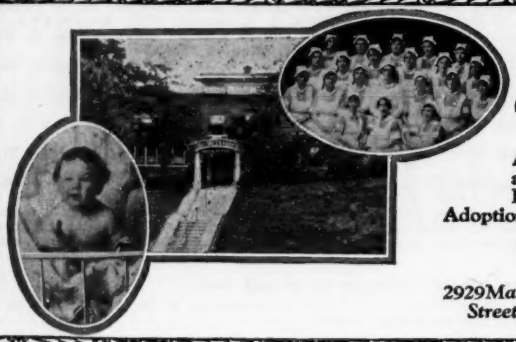

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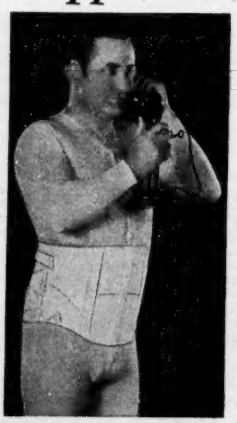

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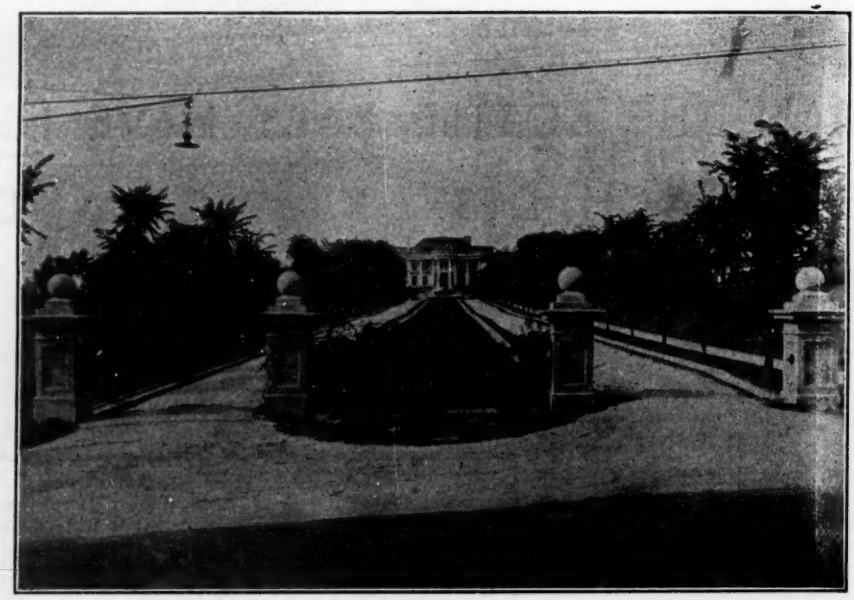

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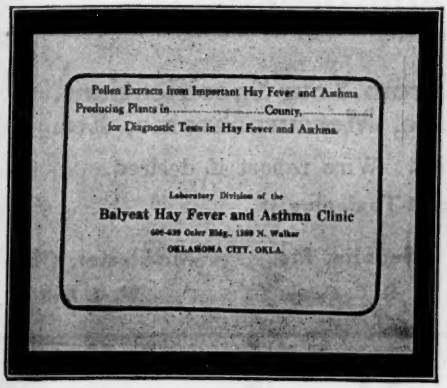
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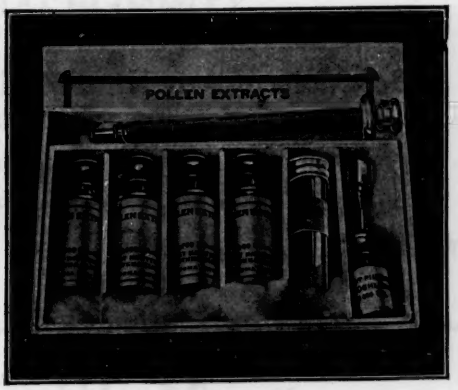

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